"PRE OPERATIVE SERUM ALBUMIN AND BODY MASS INDEX AS PREDICTORS OF POSTOPERATIVE MORBIDITY AND MORTALITY IN ELECTIVE MAJOR SURGERIES"

Dissertation submitted to

The Tamil Nadu M.G.R. Medical University

Chennai – 600032, April - 2015

In partial fulfillment of the

Regulations of the award of degree of

M.S. General Surgery

Department of General Surgery

Coimbatore Medical College Hospital

Coimbatore – 641 018

CERTIFICATE

This is to certify that this dissertation titled **"Preoperative serum albumin and Body Mass Index as predictors of postoperative morbidity and mortality in elective major surgeries"** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch – I (General Surgery) is a bonafide work done by **Dr.Raghul M,** post graduate student in General Surgery under my direct supervision and guidance during the period of September 2013 to August 2014.

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The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

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DECLARATION

I solemnly declare that the dissertation titled "**PRE OPERATIVE SERUM ALBUMIN AND BODY MASS INDEX AS PREDICTORS OF POSTOPERATIVE MORBIDITY AND MORTALITY IN ELECTIVE MAJOR SURGERIES"** was done by me at Coimbatore Medical College Hospital, Coimbatore-641 018 during the period of my post graduate study for M.S.Degree Branch – I (General Surgery) from 2012 to 2015.

This dissertation is submitted to the Tamil Nadu Dr. M. G. R. Medical University towards the partial fulfillment of the University regulations for the award of M.S. Degree in General Surgery (Branch I).

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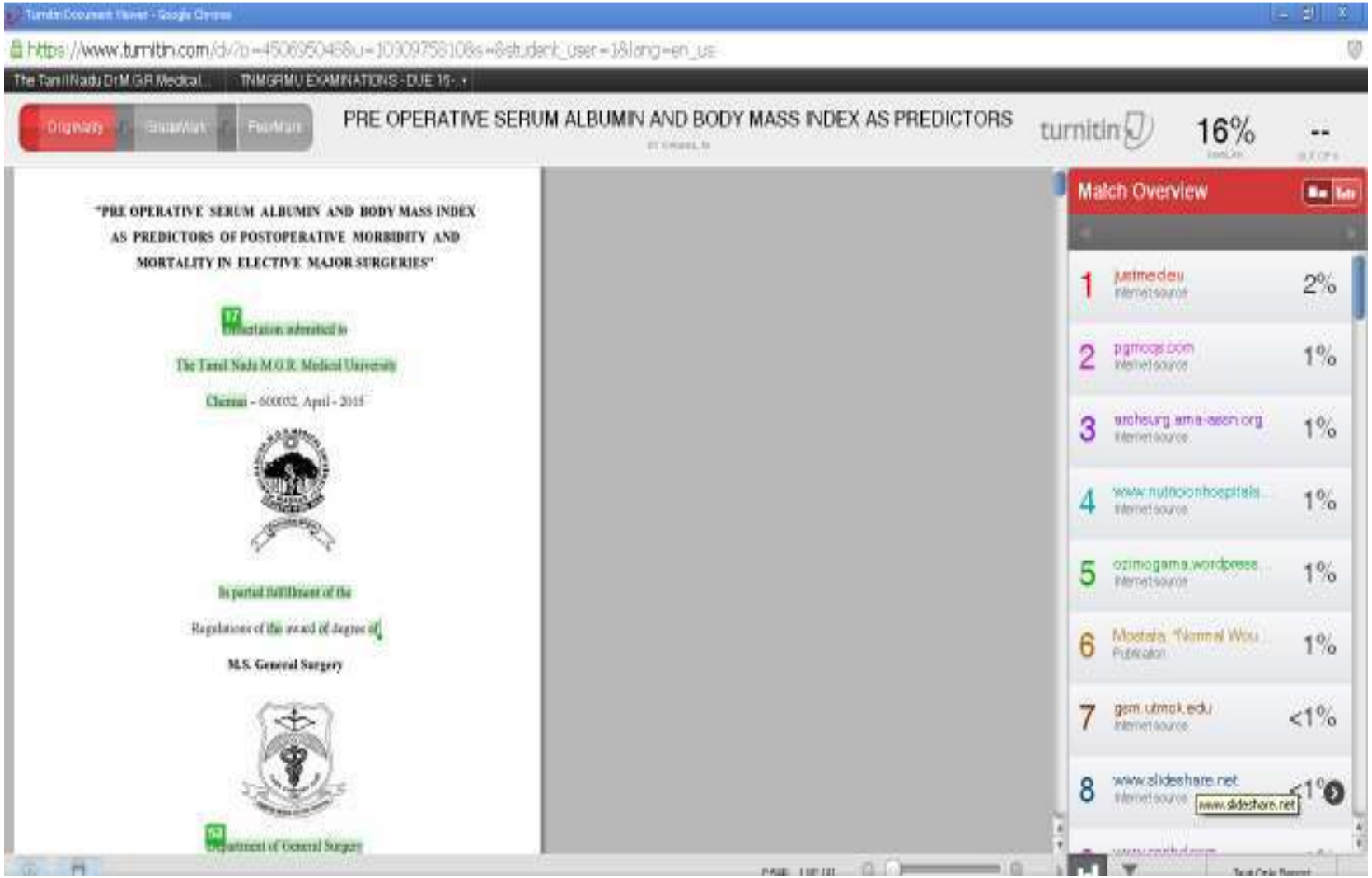
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INTRODUCTION

Wound healing requires energy and is a catabolic process. Patients who are severely malnourished demonstrate impaired wound healing and predisposition to infection. They also suffer deficient immune mechanisms. The catabolic effects of disease or injury can be reversed by adequate nutritional support. The degree of malnutrition is estimated on the basis of weight loss during the past 6 months, physical findings and plasma protein assessment.

Patient outcome can be predicted by a variety of valuable nutritional indices by means of risk stratification and objective comparison among patients but when used alone there is no consensus on the best method for assessing the nutritional status. Serum albumin level is the most readily available and clinically useful parameter. A serum albumin level greater then 3.5g/dl suggests adequate protein stores. A serum albumin level less then 3.5g/dl raises concern for potential surgical complications.

A body mass index of $19kg/m^2$ - $25kg/m^2$ for an average adult suggests a normal nutritional status. A BMI of less than $18kg/m^2$ suggests potential surgical complication.

This study therefore aims at correlating preoperative serum albumin and Body Mass Index as predictors of postoperative morbidity and mortality in elective major surgeries.

Serum albumin levels are to be measured along with BMI for all patients who are admitted for elective major abdominal surgeries in the Department of General Surgery, Coimbatore Medical College Hospital. Children < 12 yrs, patients having icterus, severe anemia (Hb < 7 gm/dl), diabetes mellitus, chronic renal disease, chronic liver disease were excluded from the study and so also patients on long term steroids or chemotherapy.

The patients are to be followed up after surgery and watched for complications like wound gaping, seroma formation, wound infection, flap necrosis, fistula formation etc. during the postoperative period.

AIMS AND OBECTIVES

- To validate preoperative serum albumin and Body Mass Index as predictors of postoperative morbidity and mortality in elective major surgeries.

REVIEW OF LITERATURE

NUTRITION IN SURGICAL PATIENT

The primary aim of providing adequate nutrition in patients undergoin surgery is to prevent or reverse the catabolic effects of disease or injury. The efficacy of nutritional regimens has been determined by several important biological parameters but the ultimate substantiation for nutritional support in surgical patients should be improvement in clinical outcome and restoration of function.[5]

The incidence of post operative morbidities, such as intra –abdominal abscess, ileus and anastomotic leakage may be brought down by the use of preoperative nutritional support. Patients with mild to moderate malnutrition need not be supplemented with preoperative nutritional support.[2]

Protein depletion results in delayed wound healing.

Malnutrition leads to significant mortalities postoperatively and threefold increase in the post operative infection rate. Current indication for nutritional support before elective surgeries include a history of weight loss in excess of 10% of body weight or an anticipated prolonged post operative period of recovery during which the patient will be nil per oral.[7]

The outcome of protein calorie malnutrition include a reduction in lean muscle mass, alteration in respiratory mechanics, impaired immune function and intestinal atrophy. These changes result in increased

postoperative morbidity due to impaired wound healing and predisposition to infection.[7]

NUTRITIONAL ASSESSMENT:

A comprehensive nutritional assessment comprises of the initial history, physical examination and laboratory testing to provide a snapshot of the patients recent nutritional health.

1) History:

The underlying disease or a history of recent weight loss suggests the possibility of malnutrition. Anorexia, nausea, gastroesophageal reflux, vomiting, dysphagia, or a history of generalized muscle weakness should be investigated for further evaluation. Recent weight loss (5% in the last month or 10% over 6 months) or a current body weight of 80 to 85% (or less) of ideal body weight implies significant malnutrition.

2) Physical Examination:

Includes muscle wasting (thenar and temporal muscles), flabby or loose skin (loss of subcutaneous fat), and peripheral edema and/or ascites (hypoproteinemia). Hepatomegaly, glossitis, pallor, skin rash, hair changes, gingival lesions and dementia are some of the subtle findings of nutritional deficiency .

3) Anthropometric measurements:

Anthropometry is the study that deals with the science of evaluating body size, weight and proportions. The body muscle mass (somatic protein) and fat reserve are reflected by measurements like MHMC (mid humeral muscle circumference) and TSF (triceps skin fold thickness) respectively.

Various body compartments (mineral, fat, lean muscle mass) can be assessed by DEXA (Dual-energy X Ray absorptiometry) and it is made available in hospitals. Measurement of weight and height with calculation of BMI can be easily performed in the bedside or at the clinic. BMI is considered to be the most reliable indicator. These values determine the patients fat reserve with visceral and somatic protein mass.[7]

4. Laboratory tests that interpret malnutrition correlate with perioperative morbidity and mortality.

- Serum albumin of less than 3.5 g% in a clinically stable, hydrated patient indicate malnutrition. In dehydrated patients, serum albumin levels are normal inspite of malnutrition. Extravascular compartment contains two thirds of the body albumin stores, remaining one third in the circulation. Plasma levels of albumin are lowered by trauma, sepsis and malignant diseases (catabolic states).

  Serum albumin has 14 -20 days as its half life.

- Serum prealbumin indicates the acute change in nutritional status. It has a half life of 2-3 days.

  10-17 mg% - mild depletion

  5-10 mg% - moderate depletion

  <5mg% - severe depletion

- Serum transferrin of less than 200mg% suggests malnutrition. It has 8 -10 days as its half life.

Malnutrition alters immune function. It can be assessed by

- Delayed type hypersensitivity
- Total lymphocyte count (TLC) that is calculated by the formula

  TLC = %lymphocyte x WBC /100

  1500 – 1800 cells/mm^3–mild depletion

  900 – 1500 cells/mm^3– moderate depletion

  <900 cells/mm^3– severe depletion

NUTRITIONAL INDICES:

They provide a means of objective comparison and risk stratification among patients. They aid surgeons in determining the correct timing for intervention and the progress being made towards the goal of adequate nourishment.[7]

BODY MASS INDEX: (Quetelet's Index)

The Belgian Adolphe Quetelet invented this measure between 1830 and 1850 during the course of developing " Social Physics" [11] It is a statistical measure of the weight of a person scaled in relation to height. BMI is defined as an individual's body weight divided by the square of their height. The extent to which a persons body weight departs can be assesed by calculating BMI from what is normal or desirable for a person of his or her height. The deficiency or weight excess may, in part, be accounted for by body fat or muscle

BMI = weight(Kg) / height^2 (m)

= 703 x weight (lb)/ height^2(in)

BMI correlates with body fat.

BMI raises its advantage in yielding a better estimation of body fat than body weight. BMI correlates with morbidity unlike the ideal body weight tables that were based on mortality data. It is used for both women and men[(9)].

Mortality and morbidity is correlated with both low and high BMI. Diminished work productivity and lethargy coincides with low levels of BMI. The lowest survivable levels of BMI have been estimated to be 12-13kg/m^2, as suggested by observation in anorexia nervosa, famine and starvation or by theoretical models.

Recent studies suggested that the all cause mortality risk is higher for independently living older individuals with a BMI less than 22 kg/m^2.

Owing to low BMI and inadequate dietary intake, the functional status has been diminshed among the older individuals in the community. There are few limitations of BMI. It may underestimate body fat in persons who have lost muscle mass (elderly) and may overestimate total body fat in persons who are very muscular (athletes). It will also inaccurately reflect body fat in edematous states or in individuals who are less than 5 feet tall[12].

		BMI (kg/m2)
Underweight	Grade III	<16
	Grade II	16 – 16.99
	Grade I	17 - 18.49
Normal		18.5 24.9
Overweight		25-29.9
Obesity	Grade I	30 – 34.9
	Grade II	35 – 39.9
	Grade III	>40

Table [40].

BMI is used in children differently. Here it is compared to typical values for other children of the same age but calculated in the same way as for adults. Rather than setting thresholds for underweight and overweight, the BMI percentile allows comparison with children of the same age and sex. A BMI that is less than the 5th percentile is considered underweight, between 85th and 95th percentile at risk of being overweight and above the 95th percentile is considered overweight.[11]

PROGNOSTIC NUTRITIONAL INDEX (PNI):

It has been experimented in patients undergoing either major cancer or gastrointestinal surgery and found to identify a subset of patients at increased risk of complications with accuracy. In addition, preoperative nutritional repletion has been shown to reduce post operative morbidity in this patient group.

PNI =158 – [16.6 x Alb] – [0.78 x TSF] – [0.2 x TFN] – [5.8 x DH]

Alb – Albumin

TSF – Triceps skin fold thickness (mm)

TFN – Transferrin (mg/dl)

DH – Delayed cutaneous hypersensitivity ;

induration > 5mm =2

1 -5 mm =1

Anergy =0

PNI risk of complication

Low <40%

Intermediate 40 -49%

High >50 %

NUTRITION RISK INDEX (NRI) [14]:

The index uses serum albumin and weight loss as predictors of malnutrition and successfully stratifies perioperative morbidity and mortality. Of note, supplemental nutrition often fails to improve serum albumin levels, hence the NRI is not a tool for tracking the adequacy of nutritional support.

NRI = [15.19 x Alb] +41.7 x [actual wt 9kg)/ Ideal wt (kg)]

NRI = Well nourished >100

Mild malnutrition 97.5 -100

Moderate malnutrition 83.5 -97.5

Severe Malnutrition <83.5

Catabolic Index (CI)

CI = [24 hr urine urea nitrogen excretion in g] – [0.5 x (dietary nitrogen intake in g)]

No physiologic stress 0

Mild stress 0-5

Moderate to severe stress >5

ESTIMATION OF CALORIC REQUIREMENTS: (15)

Adequate substrates are required for healing and tissue repair. Failure to provide adequate amounts of both calorie and protein leads to further depletion of lean body mass [(3)].

A modification of Herring equation has been implicated in the calculation of Basal Energy Expenditure (BEE) :

BEE in kcal/day in,

Males : 16.4+ [13.7 x weight (kg)] + [5.0 x height(cm)] – [6.8 x age(yr)]

Females: 655 + [9.6 x weight (kg)] + [1.7 x height(cm)] – [4.7 x age(yr)]

The actual caloric requirement is obtained by multiplying BEE by a specific stress factor. Most stressed patients require 25 -30 kcal/kg/day

TEE in kcal/day (Total energy expenditure)

TEE = BEE x stress factor

Selective stress factors

- Starvation 0.8 -1.0
- Elective surgery 1.0 -1.1
- Peritonitis/other infections 1.05 -1.25
- ARDS or sepsis 1.3 - 1.35
- Cardiopulmonary disease 1.3 – 1.55
- Pancreatitis or major burns 1.3-1.8

Estimation of protein requirement:

The protein requirement differs according to the clinical condition and also the patient. The appropriate ratio of caloric: nitrogen is 150:1 (for a caloric: protein ratio of 24:1)

Clinical condition	Protein requirement (g/dl ideal body Wt/day)
Normal person	0.8
Elective hospitalization	1.00 – 1.10
Complicated post operative case, infection	1.20 – 1.40
Major trauma, sepsis, pancreatitis	1.50 – 2.50

Protein needs can also be assessed in serum albumin levels.[11]

	Albumin (g/dl)	Protein requirement (g/kg/day)
Normal nutritional status	>3.5	0.8
Mild depletion	2.8 -3.5	1.0 – 1.2
Moderate depletion	2.1 – 2.7	1.2 -1.5
Severe depletion	<2.1	1.5 – 2.0

METABOLISM OF PROTEINS, CARBOHYDRATES AND FATS:

Human body requires energy to maintain itself in a steady state. About 50% of the basal metabolic rate (BMR) represents the work of ion pumping, 30% reflects turnover of proteins and the remaining energy used for recycling of glucose, pyruvate , amino acids and lactate . Energy consumed during physical activity comprises 10 - 20% of the total in hospitalised patients and about 10-50% in normal subjects.

The increase in energy expenditure above basal is about 10% for elective surgeries, 10-30% for physical trauma and 50-80% for sepsis.

Carbohydrates, proteins and fats are the sources of metabolic energy.

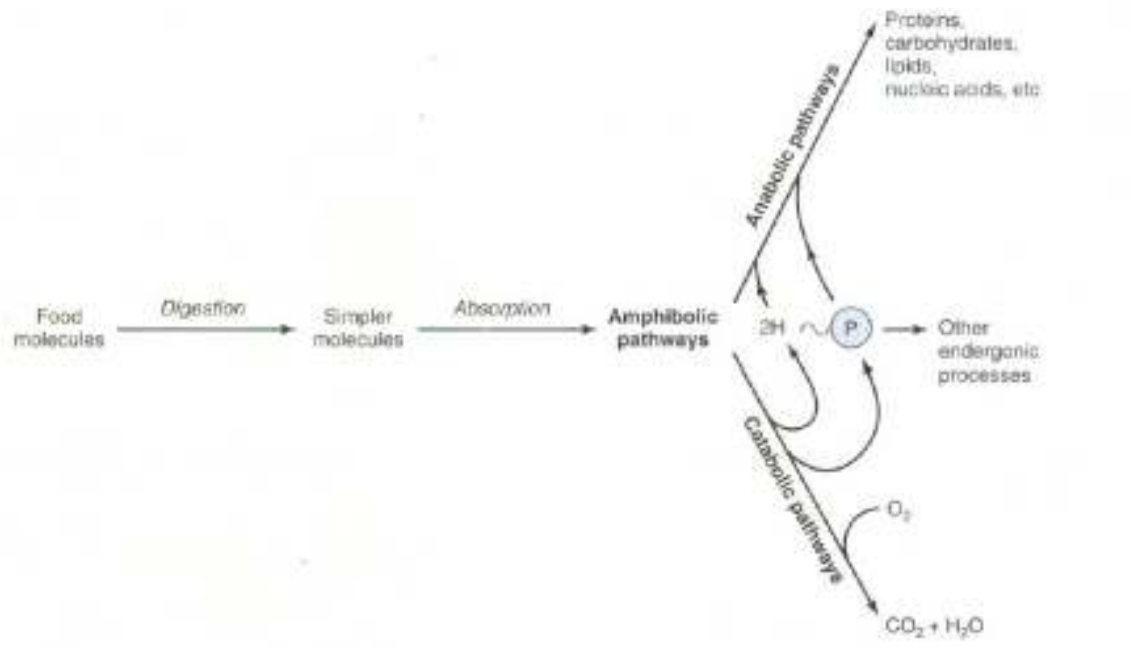


Fig.1 Metabolism of food molecules.

Carbohydrate Metabolism:

The body's primary fuel source is constituted by carbohydrates normally that accounts for 30-40% of total caloric intake. Each gram of enteric carbohydrate yields 4 kcal of energy. Parenterally administered carbohydrate (example: IV dextrose) provides 3.4 kcal per gram.

Salivary amylase initiates carbohydrate digestion and completion of absorption occurs within 1 - 1.5 m of small intestine. Starch is cleaved by salivary and pancreatic amylases into oligosaccharides. These molecules are hydrolyzed and transported by surface oligocassharides.

More than 75% of carbohydrates that are ingested are broken down and absorbed as glucose.Insulin secretion from pancreatic beta cells is stimulated by hyperglycemia. Insulin then influences protein synthesis.

A minimum intake of 400 kcal of carbohydrate per day reduces protein breakdown, especially after adaptation to starvation. Wound healing requires glucose as a major component, but excessive carbohydrate intake or repletion with excess amounts of glucose can result in hepatic steatosis and neutrophil dysfunction.

Excess glucose from refeeding, as reflected by RQ >1.0,occurs in conditions such as thermogenesis, glucosuria and conversion to fat (lipogenesis).

Excessive glucose administration is deleterious in patients with suboptimal pulmonary function because of its elevated carbon-di-oxide production.

It results in hyperglycemia, which may contribute to increased risk of infection and immune suppression.

As a result of carbohydrate deficiency breakdown of muscle tissues will occur. Fatiguability will set in as deficiency evolves. Cerebral metabolism diminishes and thinking capacity is hindered.

Deficiency also causes ketosis and ultimately weight loss which on a long run can cause acidosis.

In addition patient may have nausea, mood swings, dizziness, weakness and depression.

Hence adequate dietary carbohydrate is mandatory.

The following flow chart gives an over view of major pathways and their corresponding endproducts.

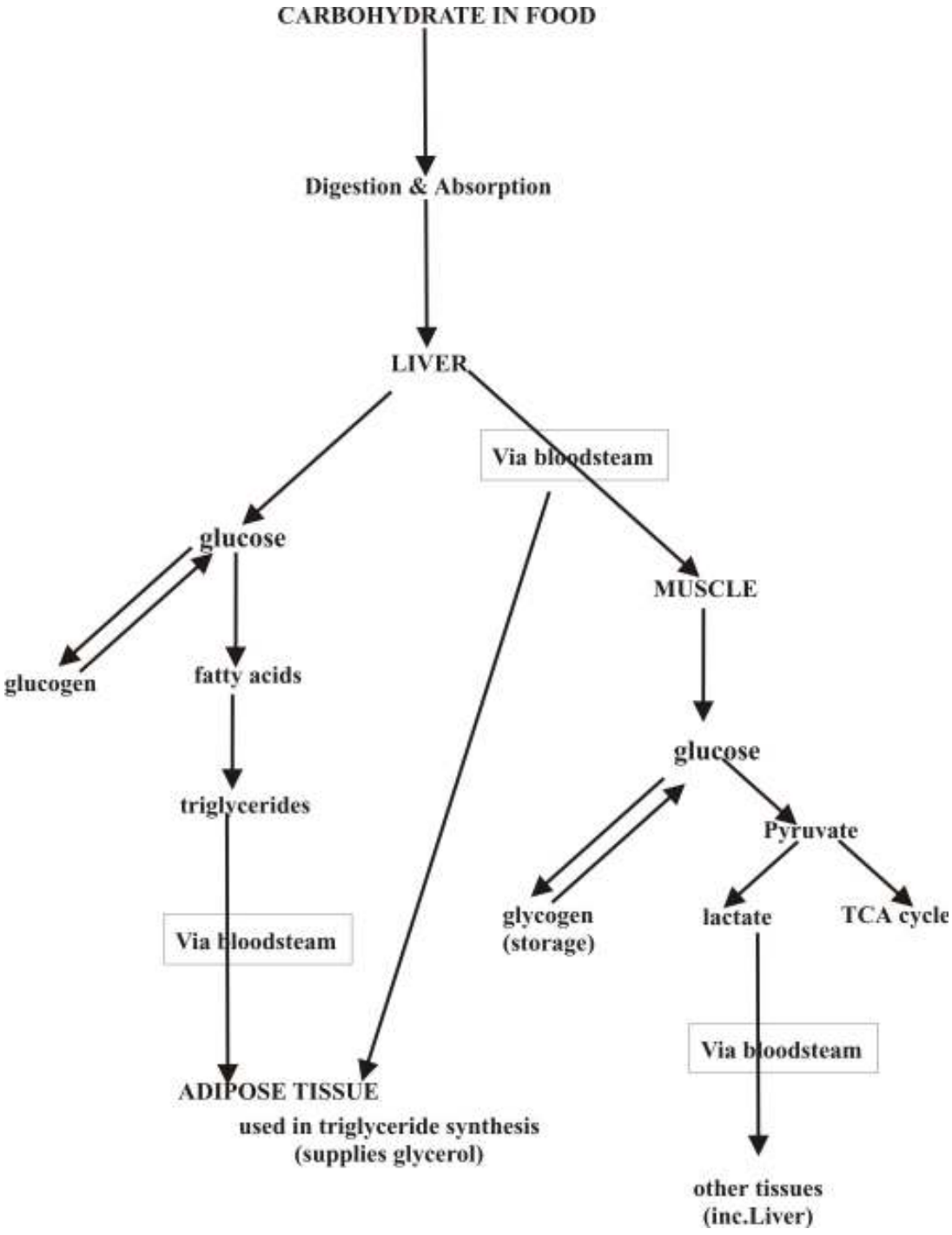


Fig.2: Carbohydrate metabolism - an overview showing major pathways and endproducts.

Protein metabolism:

Amino acids comprise proteins their and metabolism produces 4 Kcal/gm of protein. Single aminoacids and dipeptides are produced by protein digestion that are absorbed by the gastrointestinal tract. The process of digestion is initiated by gastric pepsin. Enterokinase, that are the principal effectors of protein degradation are found throughout the duodenal mucosa and activate pancreatic proteases. Once digested, almost 50% of protein is absorbed in duodenum and complete protein absorption occurs in the mid jejunum.

A 70 kg person has a total body protein of 10 - 11 kg that is concentrated predominately in skeletal muscle. Daily protein turnover is 3 % of total body protein that accounts to about 250-300 g.

The daily protein requirement in healthy adults is 0.8 g/kg body weight. Protein synthesis or breakdown can be determined by measuring the nitrogen balance.Nitrogen of 1 g accounts to about 6.25g of protein intake.

Nitrogen (balance) =Nitrogen (intake) – Nitrogen (output)

Nitrogen (intake) = g protein (intake)/6.25

Nitrogen (output) = (UUNxVol) +3

Where, UUN is urine urea nitrogen

vol is volume of urine produced over time of measurement. [33]

There are 20 amino acids that are divided into essential amino acids and non essential amino acids, that depends on whether the synthesis is de novo. Major role of amino acids are:

- Protein synthesis and recycling.
- Energy generation through catabolic reactions and carbon-di-oxide production.
- Incorporation of nitrogen into non essential amino acids and nucleotides.
- Transport and storage of small molecules and ions[18]

Plasma aminoacid accumulation is regulated primarily by the hepatic metabolism of absorbed aminoacids. Urinary excretion amounts to about 10-15g of basal nitrogen loss. A decrease of protein turnover occurs as age advances with 25g/kg/day in the neonate to 3g/kg/day in the adult

Glutamine:

It is a non essential amino acid that plays a vital role in the metabolically stressed patient. It is considered to be a crucial respiratory fuel for enterocytes. The decline of glutamine associated with injury or stress exceeds that of any other amino acids and persists during recovery even after the concentration of other amino acids has normalized. It is essential for lymphocyte production and macrophage function.

Arginine:

It is a substrate for the urea cycle and nitric oxide production. It improves nitrogen balance and wound healing, stimulates T-cell response and reduces the incidence of infective complications. It also improves albumin synthesis.

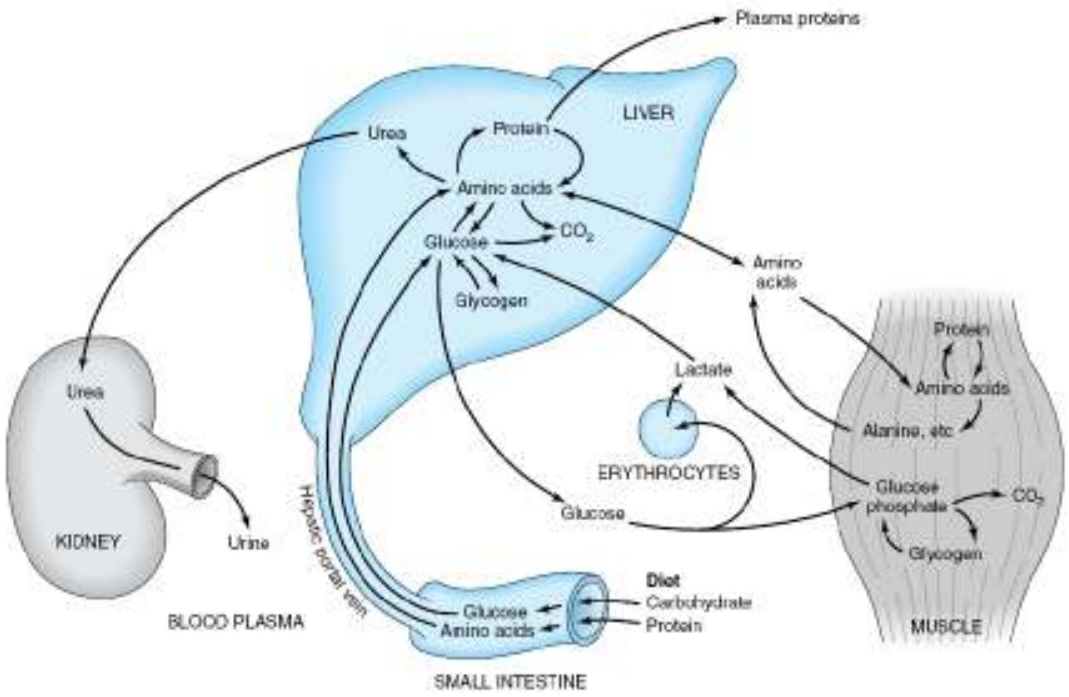


Fig.3:Overview of protein metabolism showing major pathways and endproducts

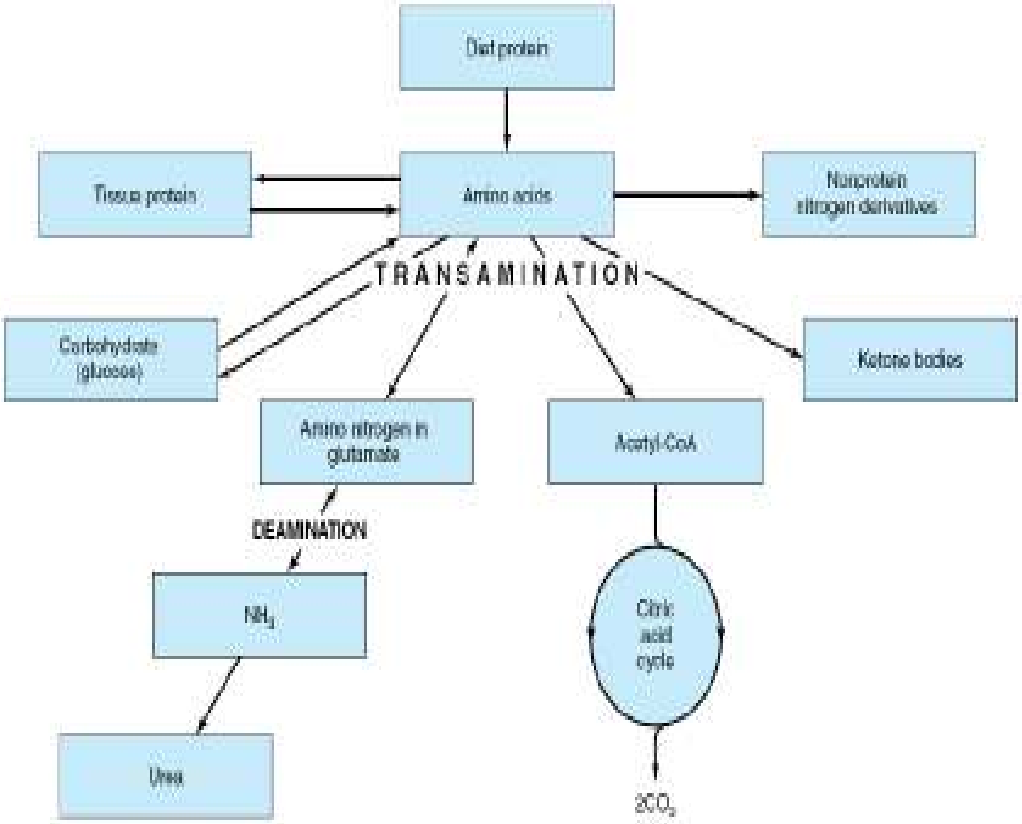


Fig.4:Overview of amino acid metabolism and major pathways.

Lipid metabolism:

About 25 - 40 % of caloric intake in a typical diet is constituted by lipids. 9 kcal of energy is obtained from each gram of lipid.A functional jejunum and ileum along with co-ordination between pancreatic and biliary secretion is required for the complex digestion of lipid molecules.

Duodenal introduction of fat results in secretion of secretin and cholecystokinin and these are essential for gall bladder contraction and pancreatic enzyme release, respectively.

The hydrolysis of triglycerides to two fatty acids and a monoglyceride by lipase is falicitated by the alkaline environment of the duodenum. Emulsification is due to bile salts. Micelle formation is the most important step in lipid absorption, which helps absorption of fats across the mucosal barrier. The bile salt pool is maintained through reabsorption of bile salts and increased hepatic cholesterol synthesis.

Increased hepatic synthesis of cholesterol compensates the intestinal bile salt losses. Bile salt pool would get depleted and fat malabsorption occurs due to major ileal resection. Glucagon , catecholamines, steroids stimulates lipolysis that is inhibited by insulin. Dramatic lipolysis occurs due to stress. The cell membrane integrity is maintained by essential fatty acids linolenic acid and linoleic acid. The sole precursors of eicosanoid production are dietary fats. Generalized scaling rash , bony changes and hepatic steatosis occurs due its clinical deficiency.

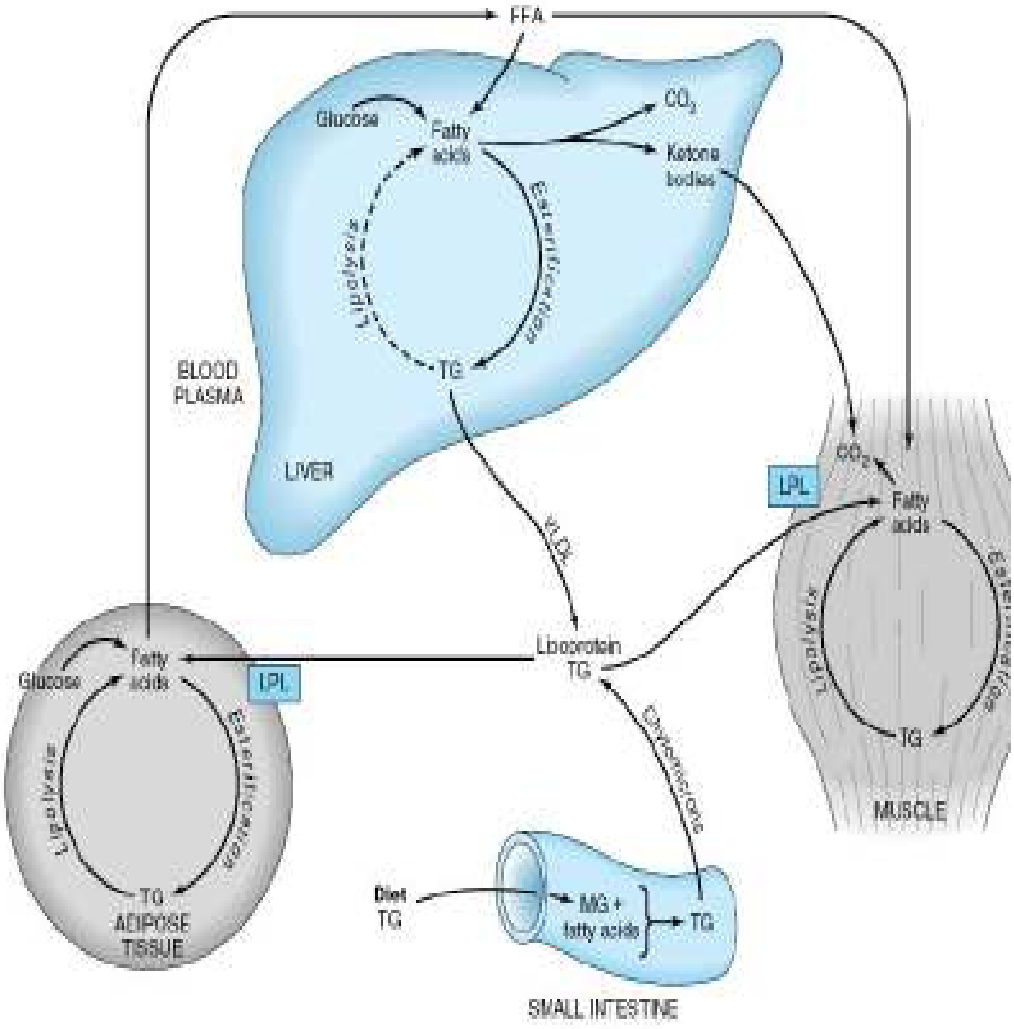


Fig.5:Overview of fat metabolism and major pathways.

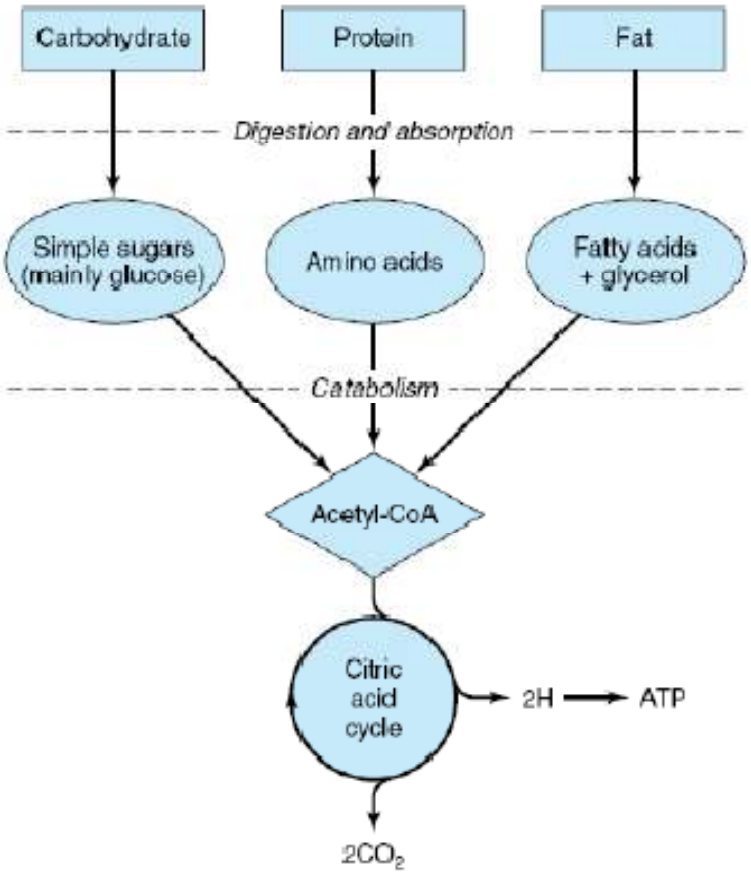


Fig. 6: Outline of the pathways for the catabolism of dietary carbohydrate, protein, and fat. All the pathways lead to the production of acetyl-CoA, which is oxidized in the citric acid cycle, ultimately yielding ATP in the process of oxidative phosphorylation.

STRESS METABOLISM:

Physiologic process, immunocompetence, wound healing and recovery from critical illness all depend upon adequate nutrient intake.

STARVATION:

Hepatic glycogen is depleted after an overnight fasting due to decreased plasma insulin and a rise in glucagon levels. After a day's fast , carbohydrate stores are depleted. Muscle glycogen is used after hepatic glycogen . Hepatic glucose metabolism must satisfy the energy demands of the hematopoietic and the central nervous system particularly the brain, which is dependant on glucose oxidation during acute starvation. Within approximately 10 days of starvation, the brain adapts to use fat as its fuel source.

Thereafter brain relies on ketoacids produced by the liver. This has a protein sparing effect. Relative protein preservation , changes in the fuel consumption , and a decrease in basal expenditure of energy are the adaptive changes to starvation (upto 30%).

Physiologic stress:

Sepsis, trauma and major operations resulting in interaction of endocrine and metabolic responses are divided into three phases

1) Catabolic phase:

There is a dramatic increase in the metabolic demand after a major surgery that is reflected by an increase in urinary nitrogen excretion. Following a major surgery , depletion of proteins initially occurs because patients commonly have an elevated basal metabolic rate and they are kept nil per oral. Physiological stress leads to hormonal responses that includes elevation in the serum glucagon , glucocorticoids and catecholamine levels.

2) Early Anabolic phase: (Cortical withdrawal phase)

Since it marks the shift from catabolism to anabolism, it ranges from several weeks to few months. A progressive gain in weight and muscle strength occurs during this phase due to positive nitrogen balance. The amount of energy lost in catabolic phase equals the amount of nitrogen gained.

3) Late Anabolic phase:

This refers to the last period of recovery that lasts for months to several weeks. Nitrogen balance equals as adipose tissue stores are lost gradually.

Following elective operation, neural impulses carried via spinothalamic pathways activate the brain stem and thalamic and cortical centers which stimulate the hypothalamus. Hypothalamic stimulation

triggers combined neural and endocrine discharges. Nor epinephrine is released from sympathetic nerve endings. Epinepheine from the adrenal medulla, Aldosterone from adrenal cortex, ADHfrom posterior pituitary, insulin and glucagon from the pancreas and ACTH, TSH and GH from the anterior pituitary. These hormones produce secondary elevations of cortisol, thyroid hormone and somatomedins. The effects of the heightened neuroendocrine secretion include

a) peripheral lipolysis from the synergistic activation of hormone sensitive lipase by glucagon, epinephrine, cortisol and thyroid hormone.
b) accelerated catabolism, a rise in proteolysis stimulated by cortisol and
c) decreased peripheral glucose uptake due to insulin antagonism by GH and epinephrine.

The consequences are a marked rise in plasma concentration of free fatty acids, glycerol, glucose, lactate and amino acids.

The liver responds with an increase in substrate uptake and glucose production, as a result of glucagon stimulated glycogenolysis and enhanced gluconeogenesis induced by cortisol and glucagon.

Operation and trauma are neuroendocrine driven processes. The BEE rises by 10% in post operative patients.

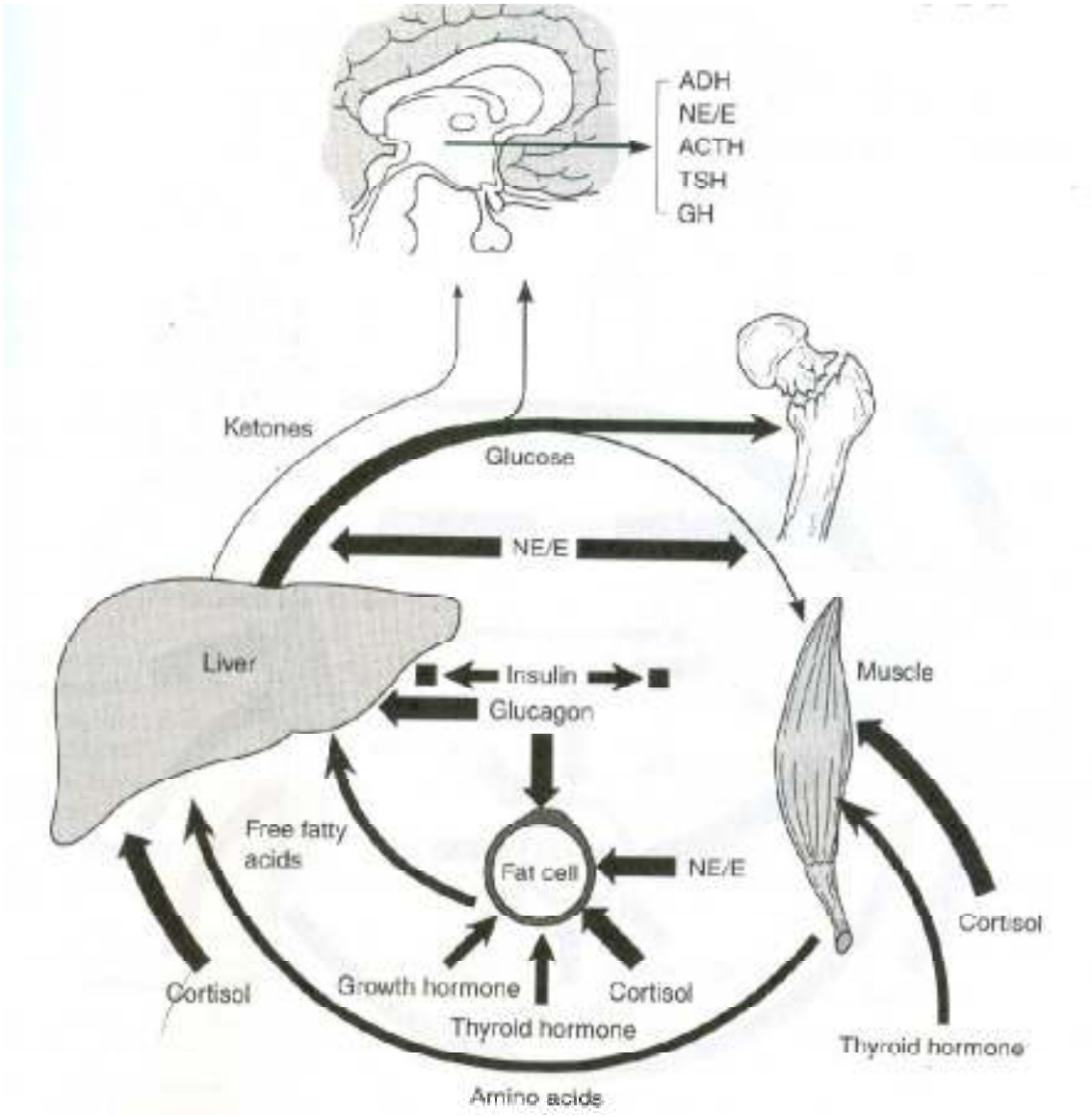


Fig.7: The metabolic response to trauma is a result of neuroendocrine stimulation, which accelerates protein breakdown, stimulates gluconeogenesis, and produce glucose intolerance.

WOUND HEALING:

Healing is a proliferative reaction that restores tissue integrity.

It involves the following processes.

1. Induction of an inflammatory process in response to the initial injury with removal of damaged and dead tissue.
2. Proliferation and migration of parenchymal and connective tissue cells.
3. Formation of new blood vessels (angiogenesis) and granulation tissue.
4. Synthesis of ECM proteins and collagen deposition.
5. Tissue remodeling.
6. Wound contraction.
7. Acquisition of wound strength.

Minimal intervention is required after a typical injury as wound healing per say is a normal orderly process.

A normal wound healing state is achieved after a variety of interventions that are required to shift and correct the wound healing process in chronic wound healing. Acute wounds are surgical or traumatic in origin. These wounds occur drastically and as a result of rapid and predictable repair process, results in closure.

Chronic wounds arise as a result of repeated insult to tissues, compromise to vasculature or chronic inflammation. They fail to go through the normal repair processes. They result in an untimely closure or improper wound closure.[13]

WOUND HEALING PHASES:

a. Inflammation and hemostasis
b. Proliferation
c. Maturation and Remodeling

The immediate response to injury is the inflammatory phase (also called reactive phase). The body's defence system aims to prevent further injury or to limit the amount of damage. The proliferative phase (also called regenerative or reparative phase) consists of re-epithelialisation, matrix synthesis and neovascularisation to relieve ischemic trauma itself . The final phase is maturational (or remodeling) phase which is defined as the period of contraction of the scar with cross linking of the collagen and loss of edema.

INFLAMATORY PHASE

Inflammation occurs as an immediate reaction of the tissue to injury. Here an attempt is made to limit damage by stopping the bleeding, sealing the surface of the wound and removing any necrotic tissue, foreign debris or bacteria present. During the inflammatory phase, increase in vascular permeability, chemotactic migration of cells, cytokine secretion and growth factor migration to the inflammatory site occurs along with the migratory cell activation.[29]

HEMOSTASIS AND INFLAMMATION

Vascular damage occurs after an acute tissue injury exposing the subendothelial collagen to platelets.This leads to activation of coagulation pathway after the process of platelet aggregation. Initially increase in vascular permeability occurs due to vasodilatation that follows intense arteriolar and capillary vasocostriction. The platelets and erythrocytes adhere to the damaged caplillary endothelium along with capillary plunging , thus ceasing hemorrhage. Architecture as of the pre existing tissues is achieved by regenerartion with absence of scar formation .Regeneration though the goal of wound healing, is found only in early embryonic development or in certain tissues such as liver and bone.

However, the accuracy of regeneration is sacrificed for the inflammatory phase in wound healing of adult humans.

PROLIFERATIVE PHASE

Wound repair begins to start as the acute responses such as hemostasis and inflammation resolves through angiogenesis, fibroplasia and epithelialisation. During this phase formation of granulation tissue takes place which consists of hyaluronic acid, fibronectin, loose collagen arrangement, macrophages, fibroblasts and the capillary bed.

ANGIOGENESIS

It is a phenomenon of new vasculature formation .It supports the healing wound environment after injury. The basement membrane of post capillary venules are degraded by the activated endothelial cells, thereby allowing the migration of cells through the gaps. Division of the endothelial cells that migrate takes place resulting in tubule or lumen formation. Eventually capillary maturation occurs due to deposition of the basement membrane.

FIBROPLASIA

In connective tissues, the resting mesenchymal cells are differentiated to form the specialised cells called fibroblasts. Following an injury, the

normal fibroblasts that are dormant and scanty divide and produce the components of extracellular matrix, after being chemoattracted to the inflammatory site. The fibroblasts' chief function is considered to be the synthesis of collagen. The time needed for undifferentiated mesencymal cells to differentiate into highly specialised fibroblasts is the delay between injury and the collagen appearance in the healing wound. This period depends on the type of tissue injured. It generally takes 3 to 5 days, known as the lag phase in the process of wound healing.

The collagen synthesis rate declines after 4th week and balances rate of collagen destruction eventually with the aid of collagenase. The wound enters the collagen maturation phase at this point . The maturation phase extends for months and years. During the maturation phase glycoprotein and mucopolysaccaride levels decrease and new capillaries revert and disappear. All these changes increase the wound strength and also alter the appearance of wound.

EPITHELIALISATION:

Re-epithelialisation of wound begins within hours after injury and takes 48 hours to complete in case of approximated incised wounds. Initially wound is quickly sealed by formation of clot and then by migration of epithelial cells across the defect. Keratinocytes are situated at basal layer of the residual epidermis and also in depths of dermal appendages lined by

epithelium. These migrate to re surface the wound. Epithelialisation is characterized by a series of changes in the wound that is detachment, migration and proliferation after which follows the differentiation and later stratification. Epithelialisation proceeds more rapidly only when the basement membrane zone is intact.

EXTRACELLULAR MATRIX

The ECM provides a scaffold for the stabilization of the physical structure of the tissues. Cells within it produces the macromolecular constituent that includes the following:

- Glycosaminoglycans, or polysaccharide chains are usually found linked covalently to protein in the form of proteoglycans.
- Fibrous proteins that includes collagen, fibronectin , elastin and laminin.

The wound matrix accumulates and changes in composition take place as healing progresses, maintaining a balance between new deposition and degradation. The provisional matrix exists as a scaffold for cellular migration and composed of fibrin, fibrinogen, fibrinonectin, and vitronectin. Glycosaminoglycans and proteoglycans which are synthesized later aid in matrix deposition and remodeling. Collagen which are the major scar proteins are the final results. [32]

WOUND CONTRACTION:

The wounds undergo certain degree of contraction. The myofibroblast is the major cell responsible in this process. These cells start appearing from day 6. Immediately after injury the wound contraction almost begins. The cellular movement with concomitant cytoskeleton reorganization is responsible for the wound contraction.

WOUND STRENGTH :

At the 1st week end, the strength of the wound is roughly 10% that of a normal unwounded skin, which quickly increases within next 4 weeks. By the end of 3rd month, a plateau accounting for about 70% to 80% of the normal unwounded skin's tensile strength, is reached. This condition may persist for life. The excess of collagen formation over collagen destruction during the 1st 2 months of healing and at later times from structural modification of collagen fibres (cross linking, increased fibre size) after cessation of collagen synthesis is found to be responsible for the recovery of tensile strength.[33]

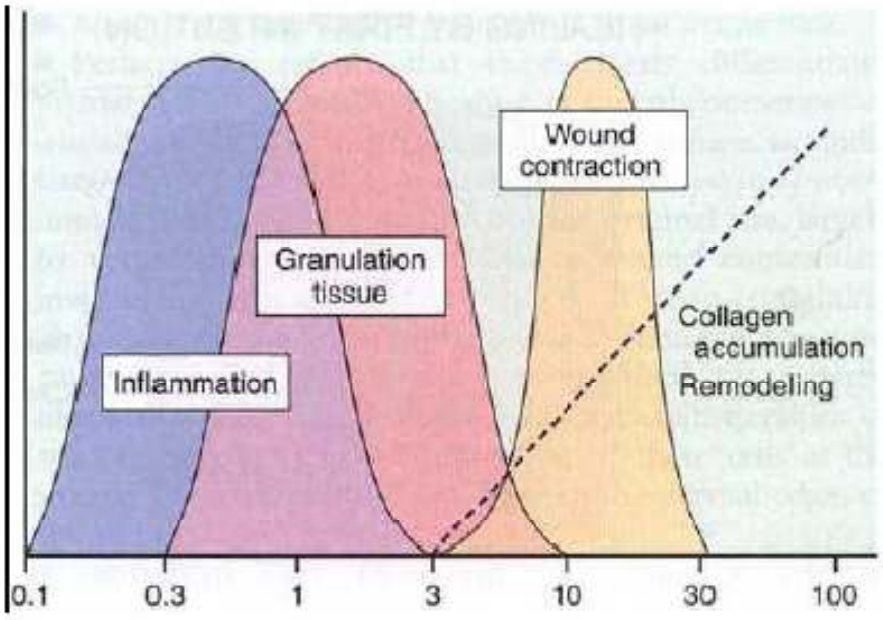
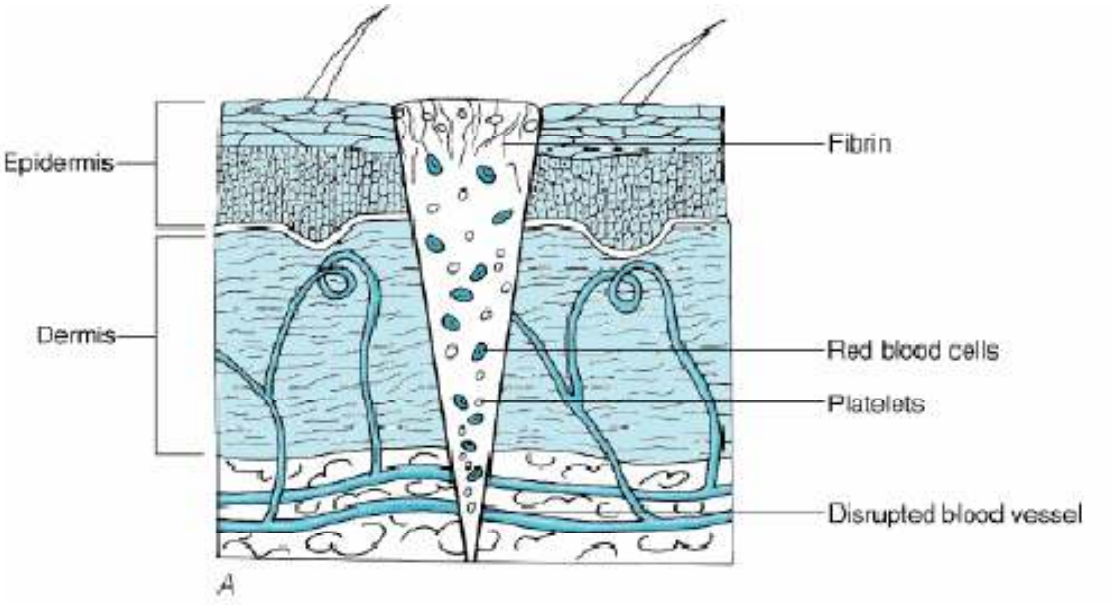
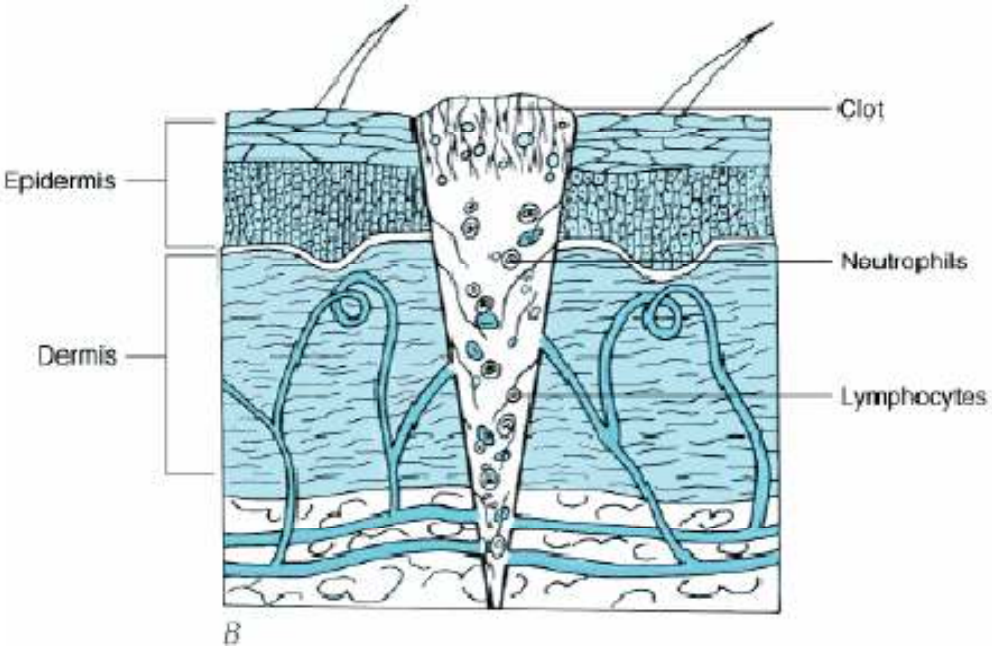


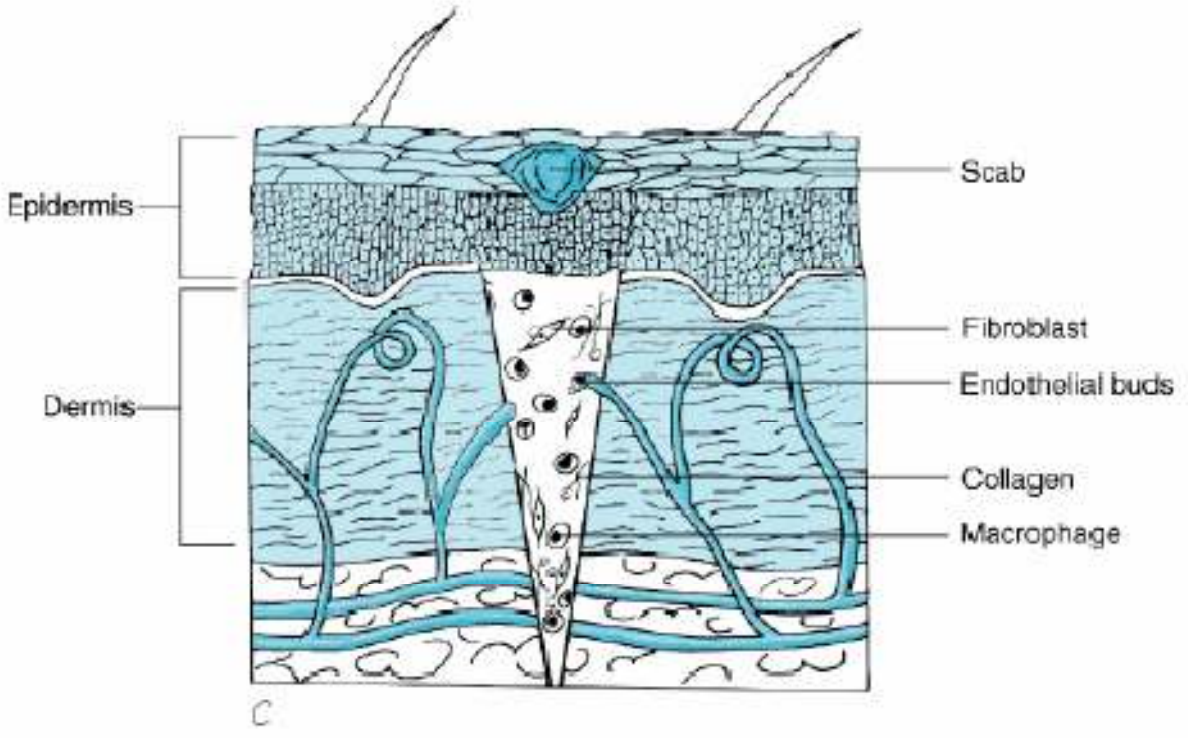
Fig.8: Phases of wound healing.[41]

This picture clearly depicts the various wound healing phases where the inflammatory phase in the earlier stages progresses to granulation tissue formation and ultimately wound contraction. Subsequently collagen accumulation and remodelling occurs.

Fig.9: The phases of wound healing viewed histologically:

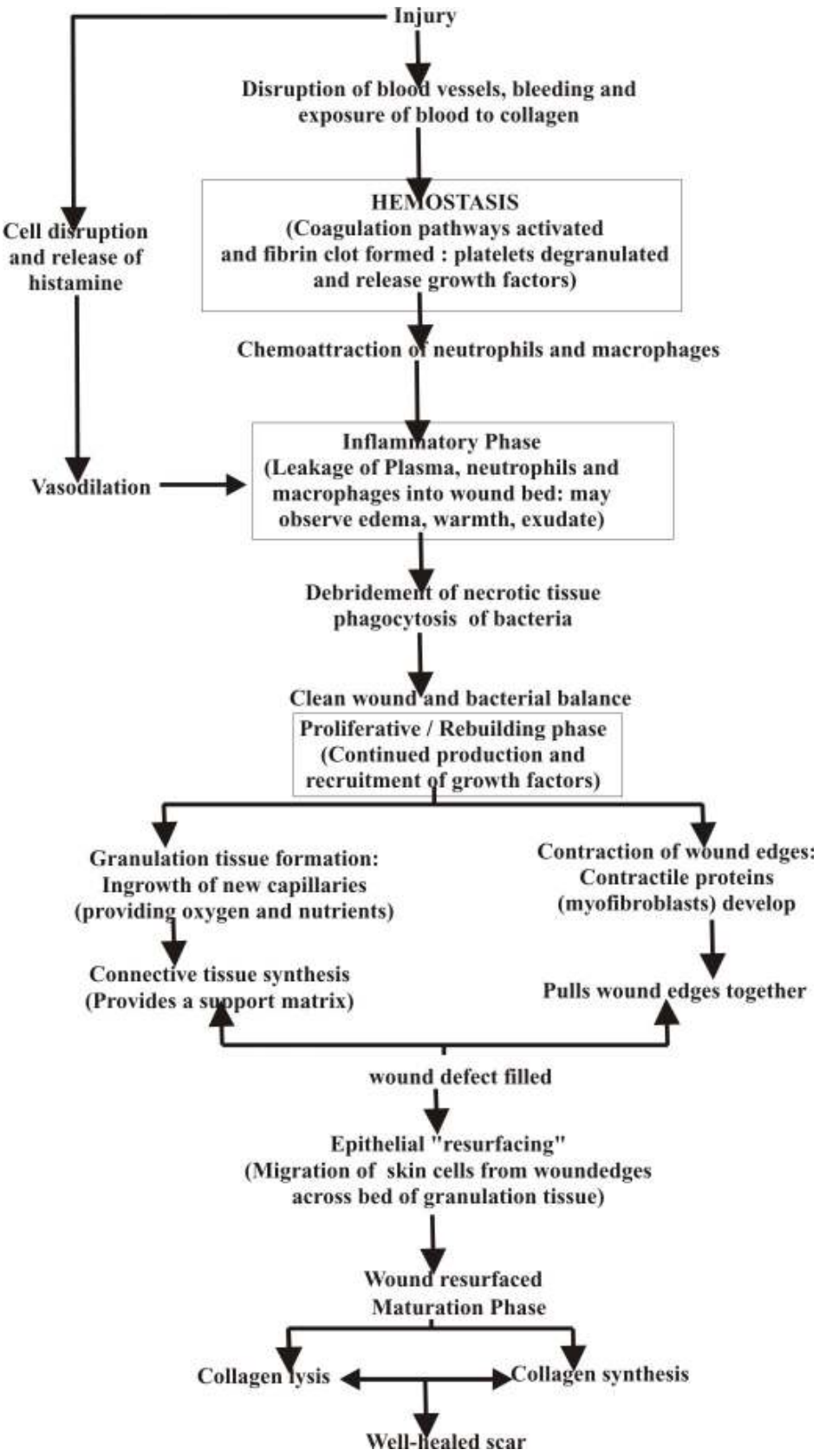






The pictorial representatives clearly depict the various histological stages where in platelets start aggregating once the subendothelial collagen matrix is exposed, with subsequent accumulation of lymphocytes and macrophages and the meshwork of collagen, fibrolasts migrate to the wound site and thus aid in healing.

Fig: 10: Cascade of events in wound repair process:



Based on timing of wound healing they are classified into:

A. Healing by primary intention

B. Healing by secondary intention

C. Healing by tertiary intention or delayed primary closure

HEALING BY PRIMARY INTENTION:

It occurs when the wound is closed by direct approximation of the wound margins or by placement of a graft or flap. Wounds that are less than 6 hrs old are considered and are less likely to develop into chronic wounds.

HEALING BY SECONDARY INTENTION:

It occurs when a wound is left open and is allowed to close by epithelialization and contraction. It is commonly used in the management of wounds that are treated beyond the initial 6 hrs.

HEALING BY TERTIARY INTENTION:

It is used for wounds that are heavily contaminated for primary closure but appears clean and well vascularised after 4-5 days of open observation so that the cutaneous edges can be approximated at that time.

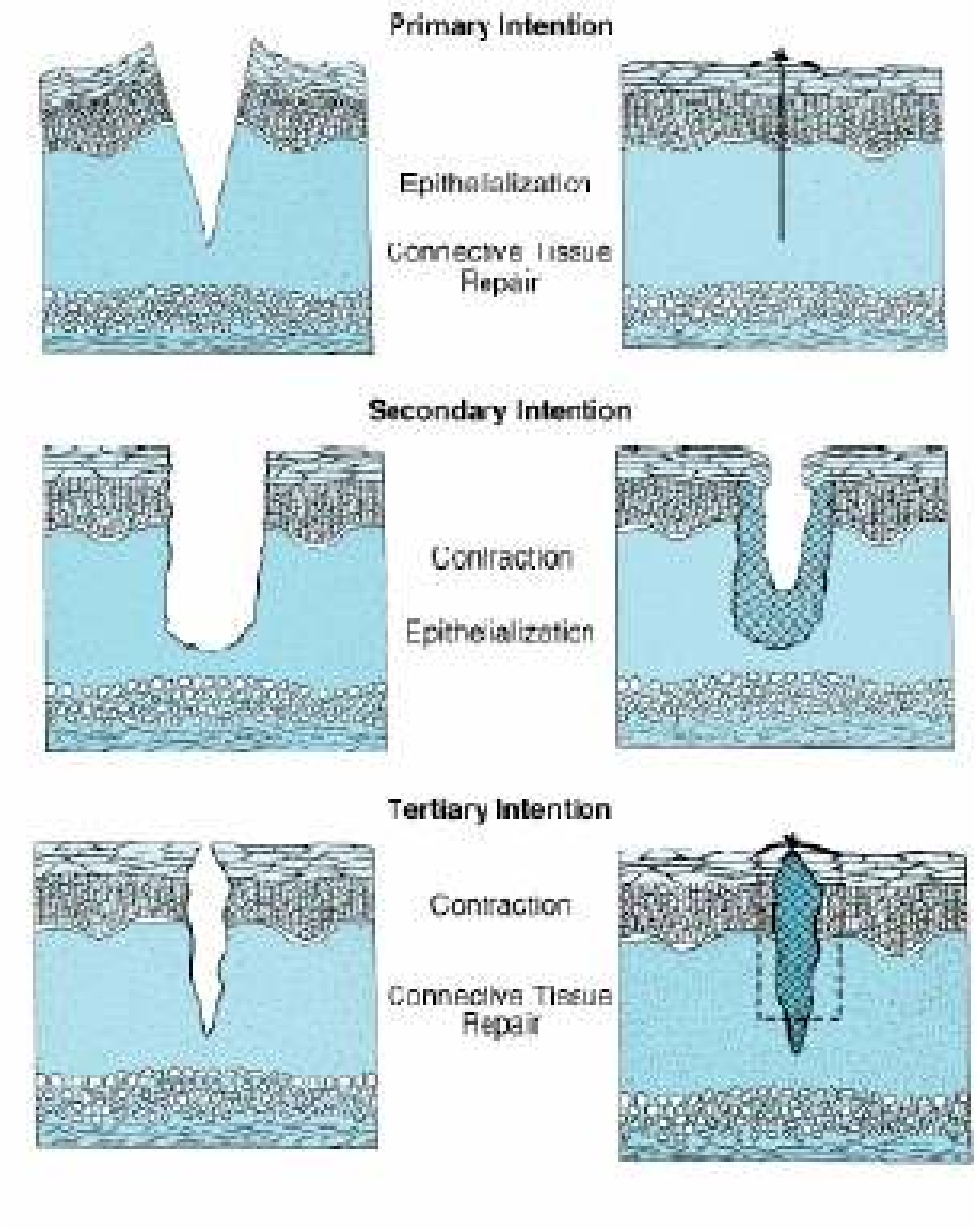


Fig.11:Classification of wound healing.

ABNORMAL WOUND HEALING:

In such a complex series of intervening events as wound healing, multiple factors can impede the outcome affect. The factors that affect the time of recovery are the amount of tissue lost or damaged, the amount of foreign material or bacterial inoculation and the length of exposure to toxins. The greater the insult, the longer the reparative process and the greater the amount of residual scar. Intrinsic factors that affect wound healing are chemotherapeutic agents, atherosclerosis, cardiac or renal failure. The blood supply in the lower extremity is the worst in the body and that on the face and hands is the best.

FACTORS THAT INHIBIT WOUND HEALING:

- **AGE** - the older the patient, the slower the healing.
- **MALNUTRITION**- Through major direct and indirect effects, the normal healing mechanism is altered by malnutrition. In order to prevent infection, adequate nutrition is necessary which aids in maintaining a competent immune system.

  Normal tensile strength and collagen framework synthesis needs iron, magnesium, zinc, vitamins A and C, calories and adequate proteins. Delay in wound healing is due to protein catabolism.

- **INFECTION -** Hypoalbuminemia predisposes to infection due to compromised immunity. Infected wounds lead to tissue destruction by prolonging the phases of inflammation, delaying collagen synthesis, preventing epithelialization and increasing the production of inflammatory mediators like cytokines.

  Presence of more than 10 bacteria signifies infection. Critical factors of infectivity are concentration, virulence, and host resistance

- **DIABETES MELLITUS (DM) -** Impaired wound healing in diabetic patients is well established. Reduced collagen synthesis and deposition and decreased tensile strength characterise the wound repair in patients with DM .

- A direct relationship between tensile strength and glycosylated hemoglobin levels have been described. The differences in wound repair is partially explained by increased levels of proteases, decreased levels of proliferative cytokines and abnormal insulin levels. DM also results in compromised perfusion, patients with DM are at high risk of microvascular diseases. Consequently the delivery of micronutrients at the capillary level is impaired and there is an increase in vascular permeability. All these factors increase infection risk and diminished support for wound healing.

- **OBESITY -** It is independently a risk factor for impaired wound healing. Adipose tissue is poorly vascularized. In addition cardiac function is frequently compromised in obese patients, further diminishing tissue perfusion. As a result infection, seroma formation, anastomotic leak and wound dehiscence are all more common among the obese population.[42]

- **MEDICATION -** Chemotherapeutic drugs and NSAIDS aree the medications compromising wound repair. Former have an impact over rapidly dividing cells. The cells more profoundly affected are fibroblasts and myofibroblasts which would result in impaired collagen synthesis and wound contraction.

- **STRESS** - It impairs wound healing as a potential co factor with its physiological and psychological means. Stimulation of sympathetic nervous system and elevation of serum corticosteroid levels leads to compromise of immune function and perfusion due to vasoconstriction as a result of stress.

- **IMMUNOSUPPRESSION-** Immunosuppression increase susceptibility to infection and retards wound healing and this is attributed primarily to impairment of the inflammatory process.

- **RADIATION THERAPY -** It causes damage to the keratinocytes and fibroblasts as well as nutrient blood vessels resulting in delayed healing .

- **CIGARETTE SMOKING -** It affects both perfusion and oxygenation.Nicotine, carbon monoxide, and hydrogen cyanide are the three byproducts of cigarette smoking . Nicotine is a potent vasoconstrictor and potentiates platelet aggregation, the oxygen saturation is lowered due to carbon monoxide and cellular oxygen transpost is depleted by hydrogen cyanide. Therefore there exists a high incidence of wound infection, dehiscence and delayed healing among smokers. [43]

- **LOCAL FACTORS -** The local factors that affect the repair process are wound bed desiccation, pH, hypothermia, excess wound fluid, and/ or heavy bacterial colonization. A moist, clean wound surface that is maintained at a temperature of about 30 degree Celsius supports wound healing to the best.

SURGICAL SITE INFECTION

These refer to the infections that are present along a surgical tract in any location after a surgical procedure. SSI is defined by the Centre for Disease Control and Prevention as the one that can occur on any post operative day ranging from 0 to 30 days to as late as 1 year in procedures such as implantation of mesh.

Classification

- Incisional superficial SSI
- Incisonal deep SSI
- Organ/space related

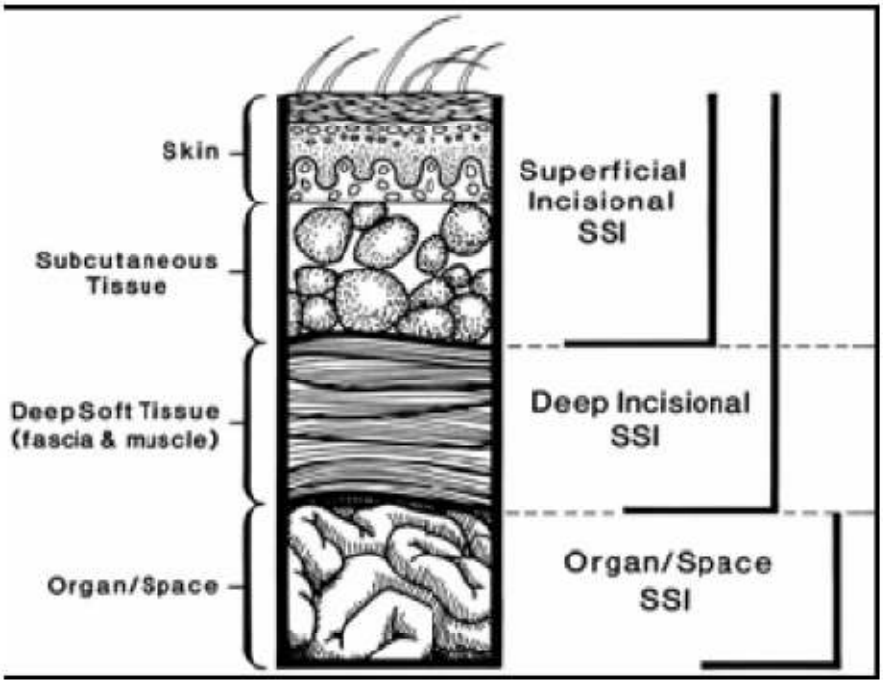


Fig.13: Types of SSI

SUPERFICIAL INCISIONAL SSI

Infection that occurs within the 30 th post operative day involving only the subcutaneous tissue and skin along with either one of the following

a. Purulent discharge
b. Isolating organisms from aseptically cultured tissue and fluid.
c. At least one sign of infection : Localised swelling, tenderness or pain, warmth and incision is deliberately opened by surgeon unless the incision is culture negative
d. Diagnosis of SSI by an attending physician or surgeon.[45]

DEEP INCISIONAL SSI

Infection occurring within 30 days of surgery or within 1 year of operation if implants are in place and infection involving deep soft tissue; and at least one of the following

a. Purulent discharge
b. Deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following symptoms; fever more than 38 degree Celsius, localized pain or tenderness unless the site is culture negative.
c. Evidence of deep infection on direct examination , during reoperation or on radiological examination.
d. Diagnosis of SSI by an attending physician or surgeon.

ORGAN/SPACE SSI:

Infection occurring within 30 days of surgery or within 1 year of operation if implants are in place and infection involving any part of anatomy that was manipulated during an operation, other than the incision and at least one of the following:

a. Purulent discharge that is placed through a stab wound into the organ space.
b. Organisms isolated from an aseptically cultured fluid or tissue. Evidence of deep infection on direct examination, during reoperation, or on radiological examination.
c. Diagnosis of SSI by an attending physician or surgeon.

About 38% of all infections in surgical patients are due to nosocomial infections among which SSI are more common. Among them incisional SSI are the most common. Organ /space related SSI account for 93% of SSI related mortality and thus have worser prognosis than incisional SSI's that account for 60 - 80% of all SSI's.

The most common pathogen in SSI is Staphylococcus aureus followed by coagulase negative staphylococcus, enterococci, and E.coli. Bacterial contamination of >10 organisms frequently causes infection whereas contamination with < 10 organisms usually does not.

Synthetic Hernia Mesh

Mesh is either synthetic – a knitted material or non-knitted sheet. It may be absorbable, or non-absorbable or a combination of both. Non-absorbable mesh, which is mostly synthetic, will stay in the body indefinitely. It is a permanent implant and difficult to remove.

Animal-Derived Hernia Mesh

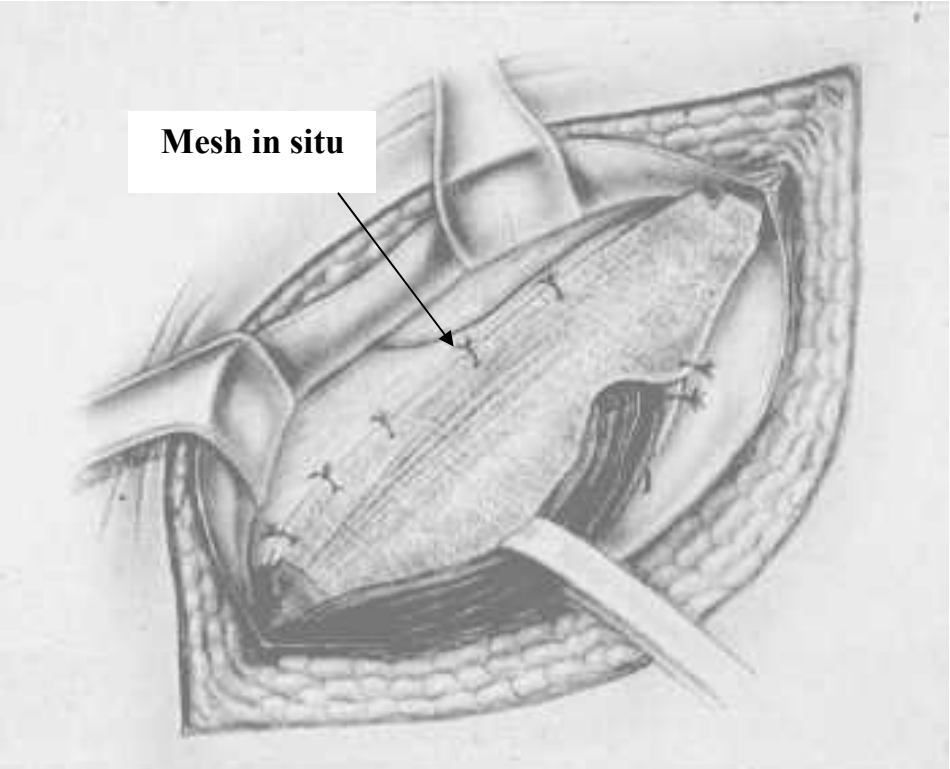
Animal-derived mesh can be made from animal tissues, such as the intestine or skin of a pig (porcine) or cow (bovine) that is processed and sterilized. Animal-derived mesh is absorbable.

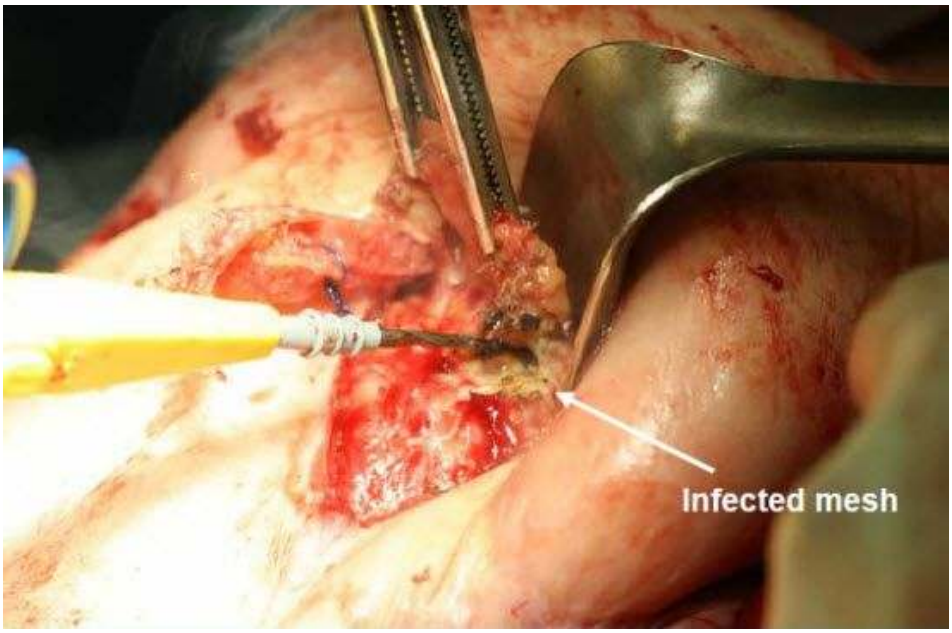
Over time, absorbable mesh will degrade and lose strength while new tissue growth takes over to strengthen the repair.

Complications associated with hernia mesh are:

* Mesh related adverse reactions
* Adhesions
* Injuries to adjacent vasculature , neuronal structures or organs
* Additional complications with or without mesh can include infection, chronic pain and hernia recurrence.

Complications can include pain, nerve entrapment, reduced movement, and a foreign object reaction that can include fever, swelling, and general malaise.





SURGICAL WOUND CLASSIFICATION ACCORDING TO DEGREE OF CONTAMIMATION :

A. Clean:

Uninfected operative wound in which no inflammation is encountered and respiratory, alimentary, genital, or infected urinary tract is not entered. Wounds are closed primarily and if necessary drained with closed drainage. Surgical wounds after blunt trauma should be included in this if they meet the criteria.

B. Clean contaminated:

An operative wound in which the respiratory, alimentary, genital or urinary tract is entered under controlled conditions and without unusual contamination.

C. Contaminated:

Open, fresh, accidental wounds. In addition operation with major break in the sterile technique or gross spillage from gastrointestinal tract and incision in which acute nonpurulent inflammation is encountered are included in this category.[48]

D. Dirty:

Old traumatic with retained and devitalized tissue and those that involve existing clinical infection or perforated viscera .This definition

suggests that the organisms causing postoperative infection were present in the operative field before the operation. The accepted range of infection rates has been 1-5% for clean, 3%-11% for clean contaminated , 10%-17% for contaminated, and >27% for dirty wounds.

Grade	Appearance
0	Normal healing
1	Normal healing with mild bruising or erythema
1a	Some bruising
1b	Considerable bruising
1c	Mild erythema
2	Erythema + other signs of inflammation
2a	At one point
2b	Around sutures
2c	Along wound
2d	Around wound
3	Clear or haemoserous discharge
3a	At one point only (<2cm)
3b	Along wound (>2cm)
3c	Large volume
3d	Prolonged (>3days)
Major complications	
4	Pus
4a	At one point only (<2cm)
4b	Along wound (>2cm)
5	Deep or serve wound infection with or without tissue breakdown; haematoma requiring aspiration

Fig.14 Southampton wound grading system:

PULMONARY COMPLICATIONS:

There are numerous factors accounting for the altered pulmonary dynamics post operatively. In nearly all patients , there is a loss of functional residual capacity.

This can arise through an array of problems, that includes abnormal distension, a painful upper abdominal incision , obesity, a strong smoking history with associated COPD, prolonged supine positioning, and fluid overload leading to pulmonary edema.

Two types of respiratory failures are commonly described. Type1 or hypoxic with a low PaO_2 and normal $PaCO_2$. Type2 failure is associated with hypercapnia and is characterized by a low PaO_2 and high $PaCO_2$.

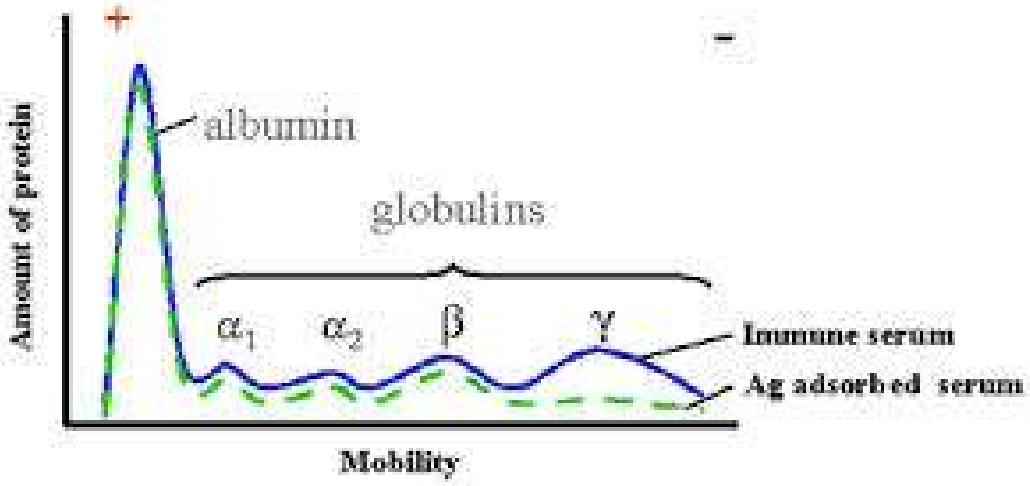
The candidates who all must be carefully screened with PFTs include patients with a history of heavy smoking, patients maintained on home oxygen, patients who are unable to walk one flight of stairs without severe respiratory compromise, patients with a previous history of major lung resection, and elderly patients who are malnourished.[14]

Several studies show that hypoalbuminemia is related to a high risk of pulmonary complications and a higher overall mortality.[8]

PLASMA PROTEINS:

The major solid components of plasma are made up of the total proteins that costitiute 7 to 7.5 g/dl. Varied types of lipoproteins and conjugated proteins also include itself along with simple proteins in the complex mixture of plasma proteins. Though under normal circumstances the amount of individual antibodies are low, there are thousands of circulating antibodies. Globulins, albumin and fibrinogen are the three major groups of antibodies.

The most common method used for analysis of plasma proteins is electrophoresis.



Functions of plasma proteins:

Function	Plasma Proteins
Antiproteases	Antichymotrypsin α_1-Antitrypsin (α_1-antiproteinase) α_2-Macroglobulin Antithrombin
Blood clotting	Various coagulation factors, fibrinogen
Enzymes	Function in blood, eg, coagulation factors, cholinesterase Leakage from cells or tissues, eg, aminotransferases
Hormones	Erythropoietin[1]
Immune defense	Immunoglobulins, complement proteins, β_2-microglobulin
Involvement in inflammatory responses	Acute phase response proteins (eg, C-reactive protein, α_1-acid glycoprotein [orosomucoid])
Oncofetal	α_1-Fetoprotein (AFP)
Transport or binding proteins	Albumin (various ligands, including bilirubin, free fatty acids, ions [Ca^{2+}], metals [eg, Cu^{2+}, Zn^{2+}], metheme, steroids, other hormones, and a variety of drugs Ceruloplasmin (contains Cu^{3+}; albumin probably more important in physiologic transport of Cu^{2+}) Corticosteroid-binding globulin (transcortin) (binds cortisol) Haptoglobin (binds extracorpuscular hemoglobin) Lipoproteins (chylomicrons, VLDL, LDL, HDL) Hemopexin (binds heme) Retinol-binding protein (binds retinol) Sex hormone–binding globulin (binds testosterone, estradiol) Thyroid-binding globulin (binds T_4, T_3) Transferrin (transport iron) Transthyretin (formerly prealbumin; binds T_4 and forms a complex with retinol-binding protein)

[1]Various other protein hormones circulate in the blood but are not usually designated as plasma proteins. Similarly, ferritin is also found in plasma in small amounts, but it too is not usually characterized as a plasma protein.

ALBUMIN:

About 60% of total plasma proteins is constituted by the major protein of human plasma - albumin. The normal serum value is between 3.5-5.5 g/dl. Molecular weight is 69kda.[15]

The total exchangeable pool of albumin is 4-5g/kg body wt; between 6-10% of the exchangeable pool is degraded per day. [16]

Human albumin does not increase the viscosity of plasma due to its ellipsoid shape and consists of 17 disulfide bonds and 585 aminoacids arranged in a single polypeptide chain. It accounts for 75-80% of human plasma 's osmotic pressure. Total protein of greater than 5 g/dl and serum albumin of greater than 2.5 g/dl is needed to produce adequate oncotic pressure.

Albumin synthesis is decreased during fasting and in cases of protein malnutrition.[16]

Albumin is an acute phase protein the concentration of which is decreased by at least 25% following injury.

Time course of changes occurs in some major acute phase proteins. C3, C3 component of complement.

(Modified and reproduced with permission, from Gitlin JD, Colten HR: Molecular biology of acute phase plasma proteins. In Pick F et al [editors].

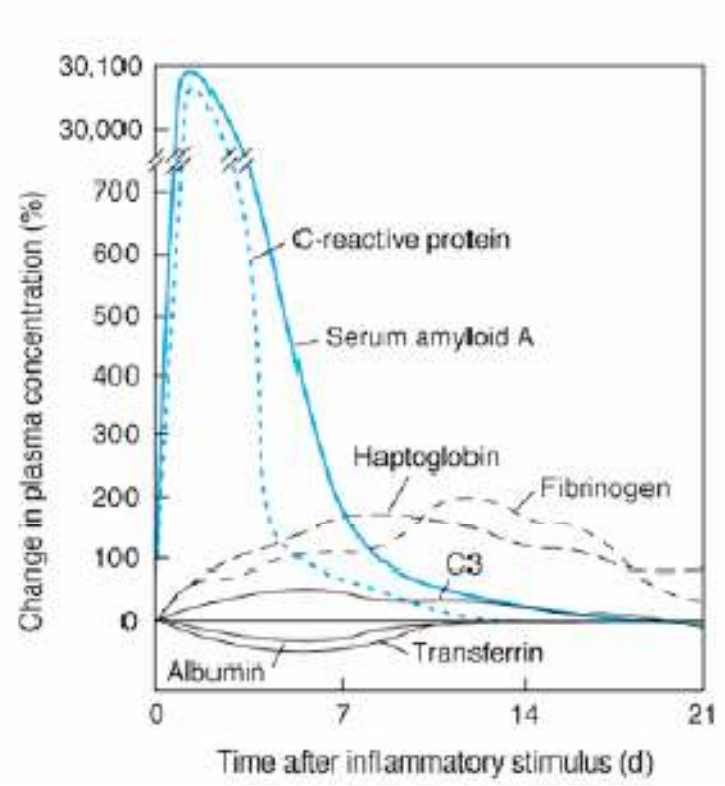


Fig.16: Time course of changes in some major acute phase proteins. C3, C3 component of complement. (Modified and reproduced with permission, from Gitlin JD, Colten HR: Molecular biology of acute phase plasma proteins. In Pick F et al [editors]. *Lymphokines*, vol 14, pages 123-153. Academic Press, 1987.)

EXTENT OF MALNUTRITION

- Normal- 3.5-5.5gm/dl
- Mild – 2.8-3.5gm/dl
- Moderate - 2.1-2.7gm/dl
- Severe- <2.1gm/dl

CAUSES OF HYPERALBUMINEMIA (>5.5g/dl)

There are no known pathological causes for a raised plasma albumin. Causes include:

a. Dehydration: this is also reflected in an increase in plasma total protein, a raised hematocrit and appropriate clinical features.
b. Venous stasis
c. Albumin infusion

CAUSES OF HYPOALBUMINEMIA (<3.5g/dl)

a. Increase in plasma H

This occurs as a part of physiological response in pregnancy. Other causes include, excessive infusion of IV fluids, water retention in SIADH, glucocorticoid deficiency.

b. Diminished synthesis-

Any cause of generalized protein malnutrition will ultimately be reflected in low plasma albumin. Causes for this include deficient diet in protein nitrogen, protein malabsorption such as coeliac disease, tropical sprue, crohn's disease, cystic fibrosis, decreased synthesis in chronic liver disease and in hereditary analbuminemia where there is a marked impairment of albumin biosynthesis with plasma levels which are typically low.

c. Increased catabolism-

This is a feature of hypercatabolic state. The important feature of hypercatabolic state is the stress related stimulation of glucocorticoid production. These hormones are known to stimulate protein catabolism. Condition such as fever, trauma, major surgery, severe sepsis, malignant disease may all be associated with varying degrees of hypoalbuminemia.

d. Losses of albumin from body-

Sites of excessive loss are GI tract, kidneys and skin.[17] Hypoalbuminemia correlates itself with immune response impairment such as activation of macrophages and formation of granuloma, decreased wound healing mechanics , and impairment of collagen meshwork synthesis at surgical wounds and anastomotic sites .This signifies the reason why infections such as pneumonia , wound infection and anastomotic site leakage were commonly found among patients with hypoalbuminemia.[37]

Gibbs et al studied the precision and reliability of estimates of the association between preoperative serum albumin concentration and surgical outcome. A total of 54,215 major non cardiac cases were studied.Serum albumin decrease from concentration > 46gm/dl to

< 21gm/dl was related with an exponential increase in mortality rates from <1% to 29% and in morbidity rates from 10% to 65%. Albumin levels were a better predictor of morbidities, particularly major infection and sepsis. Serum albumin is a better predictor of surgical outcome than many other preoperative patient characteristics. It is a relatively low cost test that should be used more frequently as a prognostic tool to detect malnutrition and risk of adverse surgical outcomes.

A retrospective study by Kudsk et al of 526 surgical patients who had preoperative serum albumin levels measured and were undergoing elective esophageal , gastric, pancreaticoduodenal or colon surgery, serum albumin levels below 3.25g/dl correlated immensely with complications, length of stay, postoperative stay and mortality.

In a study done by M.B. Badia et al of 158 patients on multivariate analysis, preoperative hypoalbuminemia was significantly associated with higher morbidity and lengthier hospital stay regardless of the type of surgery.

In a study done by Detsky et al on 202 patients who were planned for gastrointestinal surgery, several techniques of nutritional assessments were adapted to predict major post operative complications. Subjective global assessment (SGA) and albumin were both of predictive value , and combination of these variables were

useful in differentiating low risk from high risk patients. It was concluded in a study that SGA and albumin are useful "nutritional assessment techniques" for patients undergoing major gastrointestinal surgeries if the purpose of such an assessment is to predict postoperative nutrition associated complications.

A meta-analysis of cohort studies and controlled studies of hypoalbuminemia in acute illness; is there a rationale for intervention by Vincent et al[23] shows that hypoalbuminemia was a potent dose dependant , independent predictor of poor outcome. Each 10g/dl decline in serum albumin concentration significantly raised the odds of mortality by 137%, morbidity by 89%, prolongs icu and hospital stay by 28% and 71% respectively.

A serum albumin level of <2g/dl in critically ill patients has been shown to be associated with a mortality of nearly 100%. The association between hypoalbuminemia and poor outcome appeared to be independent of both nutritional status and inflammation. The complication rates may be reduced when serum albumin level attained during albumin administration exceeds 30g/dl. Complications were higher when serum albumin level was lower than 2.5g/dl in critically ill adult patients in a study by Foley et al. [24]

In a prospective randomized clinical study by Woods et al[25] done to identify the role of serum albumin concentration on length of post operative illness , the hypothesis was that patients whose albumin levels dropped below 3.5g/dl would have a more prolonged postoperative hospital course as a result of delay in return of bowel function. Increased incidence of pneumonia, wound infection, septicemia postoperatively was reported by Brown et al[26] in their study in patients with serum albumin levels < 3g/dl.

Golub et al[27] studied the effect of hypoalbuminemia (serum albumin levels <3g/dl) upon admission of patients to surgical icu due to vascular insufficiency, hip fractures, gastrointestinal bleeding , cancer, perforated viscus, intra-abdominal infection or bowel obstruction. Complications were higher in patients with hypoalbuminemia (36.9%) and mortality of (5.8%).

In a study by Beghetto et al[28] done on 434 patients who were evaluated for the accuracy of nutritional assessment tools for predicting adverse hospital outcomes, it was concluded that serum albumin level was the strongest predictive parameter for death and hospital infection (<3.5g/dl). A BMI <18.5 kg/m^2 was also associated with death and infection postoperatively and increased length of hospital stay. [60]

Liop et al [29] found that a serum albumin below 3.5g/dl at the onset of treatment was a predictor of kidney and liver failure, hospital infection, and mortality in 12 patients strata.

While assessing 6 patients submitted to elective urology and gynecology surgical procedures, Anderson et al [30] observed that low albumin had a sensitivity of 22% and a specificity of 91% in predicting hospitalization lasting more than 10 days and a sensitivity of 10% and specificity of 86% for complications.

Hirsch et al[31] in a prospective study assessed the preoperative nutritional status of surgical patients and its relation to postoperative outcomes. Preoperative nutritional assessment included anthropometry and biochemical indices. The more useful parameters were preoperative weight loss and low serum albumin levels.

Leite et al [32] in their article studied the effect of serum albumin and clinical outcome in paediatric patients undergoing cardiac surgery. A low serum albumin level (<3g/dl) was associated with a poor postoperative outcome which included increased postsurgical infection, increased mortality and longer hospital stays.

Engelman et al[33] studied the importance of BMI and serum albumin on mortality and morbidity after cardiac surgery. Serum albumin levels <2.5g/dl was associated with risk and BMI <20kg/m^2 and > 30kg/m^2 was associated with increased rates of infection.

Mullen et al [34] studied the impact of BMI on preoperative outcome in patients undergoing major intra-abdominal surgery. Being under weight was associated with higher mortality and wound infection was more common in the obese. [61]

A study by Arozullah et al [35] on the preoperative evaluation for postoperative pulmonary complications showed that low albumin levels was associated with respiratory failure and higher postoperative mortality and morbidity rates. Moreover morbidity increases exponentially as albumin levels fall below 4g/dl. Patients with >10% weight loss in 6 months prior to surgery are at increased risk for pneumonia and respiratory failure.

In a study done by Daniel et al [36] on 183,069 pts subjected to general and vascular surgeries , it was determined through a logistic regression analysis that a serum albumin <3.5g/dl and weight loss >10% was associated with cardiac complication with a significant p value(0.0001).

In a study by Varut et al [37] 244 patients subjected to rectal cancer surgery, an assessment of the role of preoperative serum albumin as a risk factor for postoperative outcome was studied. It was concluded that preoperative hypoalbuminemia (<3.5g/dl) is an independent risk factor for postoperative complications following rectal cancer surgery including mortality. Complications, time to first

bowel movement, time to first defecation, time to resumption of normal diet and length of hospital stay. Azodi[38] studied the impact of BMI and tobacco smoking on outcome after open appendicectomy. They concluded that tobacco smoking and a BMI of 27.5kg/m^2 or more were associated with more postoperative complications after open appendicectomy in patients with non-perforated appendicitis. [62]

MATERIALS AND METHODS

SOURCE OF DATA :

Patients admitted in surgical wards at Coimbatore Medical College Hospital for any major elective abdominal surgeries between September 2013 - August 2014

SAMPLE SIZE : n = 100 patients.

METHODS OF COLLECTION OF DATA :

Inclusion Criteria

- Patients admitted for any major elective abdominal surgery including malignancy under the department of surgery in Coimbatore Medical College Hospital.
- Both males and females >12 yrs are included in the study.
- Under Hernias- epigastric, incisional and Para umbilical hernias

Exclusion Criteria

- Children < 12 yrs
- Patients who have Icterus, severe anemia < 7 gm/dl, diabetes mellitus, chronic renal disease, chronic liver disease and patients on steroids or chemotherapy - both malignant and non malignant cases.
- Under hernias -Inguinal and femoral hernias.

Methods

- Details of cases will be recorded including history, clinical examination, investigations done, surgical procedures and post operative complications.
- Anthropometry – height and weight recorded.
- Follow up till discharge from the hospital.

RESULTS

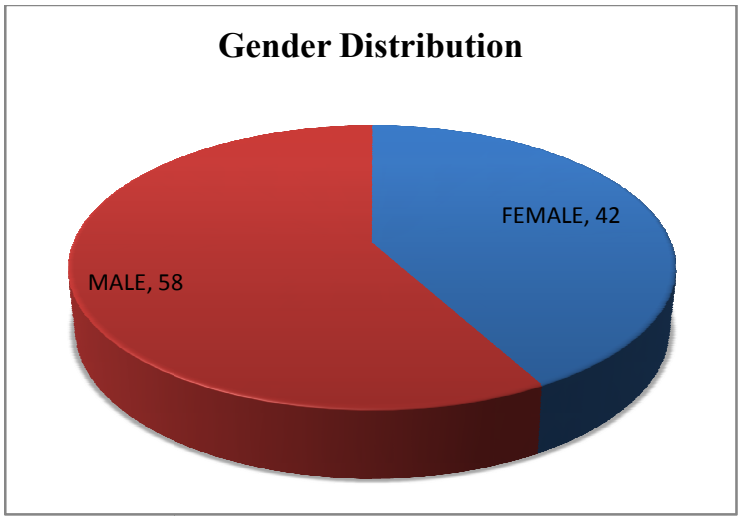
The study was conducted on 100 patients between 15 - 85 years who underwent any major elective abdominal surgery in Coimbatore Medical College Hospital, Coimbatore from September 2013 to August 2014. Among 100 patient, 42 patients developed complications and 58 had an uneventful recovery.

SEX DISTRIBUTION

Among the 100 patients under study 42 were female and 58 were male.

Frequency Table

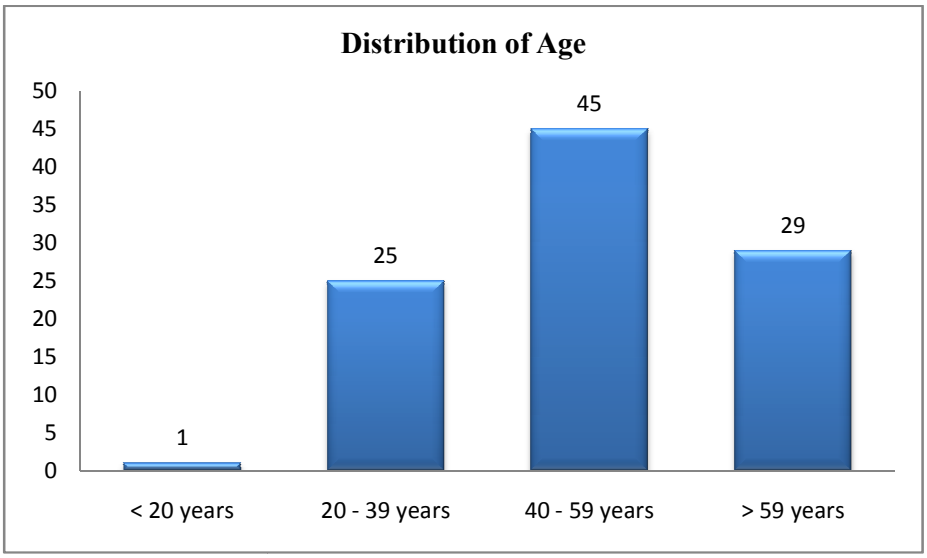
SEX					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	FEMALE	42	42.0	42.0	42.0
	MALE	58	58.0	58.0	100.0
	Total	100	100.0	100.0	



AGE WISE DISTRIBUTION OF SUBJECTS

The following is the age wise distribution of the study subjects

		Frequency	Percent	Valid Percent	Cumulative Percent
Vaild	< 20 years	1	1.0	1.0	1.0
	20 - 39 years	25	25.0	25.0	26.0
	40 - 59 years	45	45.0	45.0	71.0
	> 59 years	29	29.0	29.0	100.0
	Total	100	100.0	100.0	

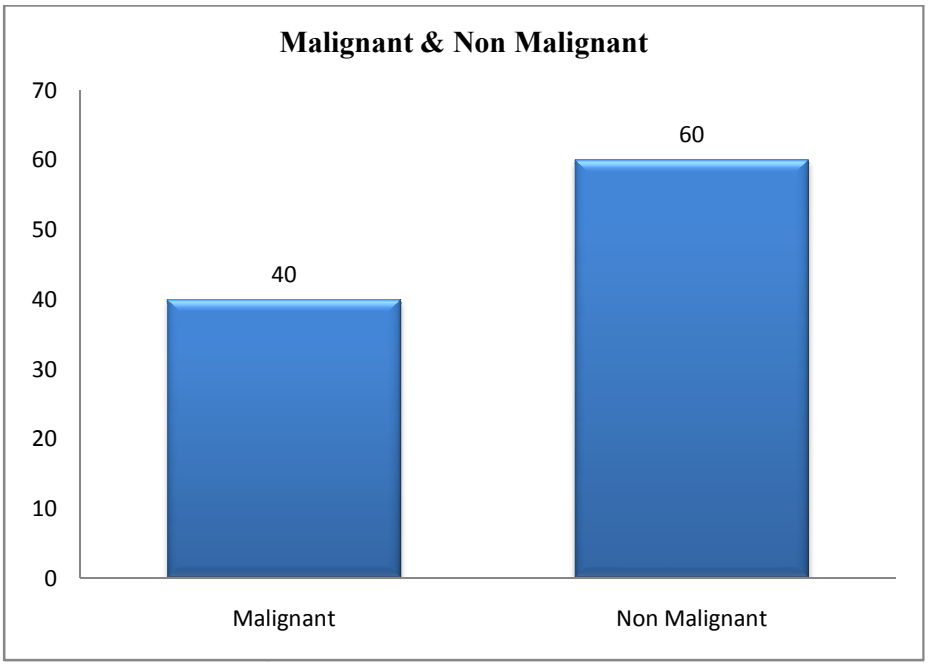


The maximum number of study subjects were in the age group of 40-59 years accounting for 45 % and there was only one subject below 20 years (15 years)

DISTRIBUTION OF MALIGNANT AND NON MALIGNANT CASES

MALIGNANT & NON MALIGNANT

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Malignant	40	40.0	40.0	40.0
	Non Malignant	60	60.0	60.0	100.0
	Total	100	100.0	100.0	

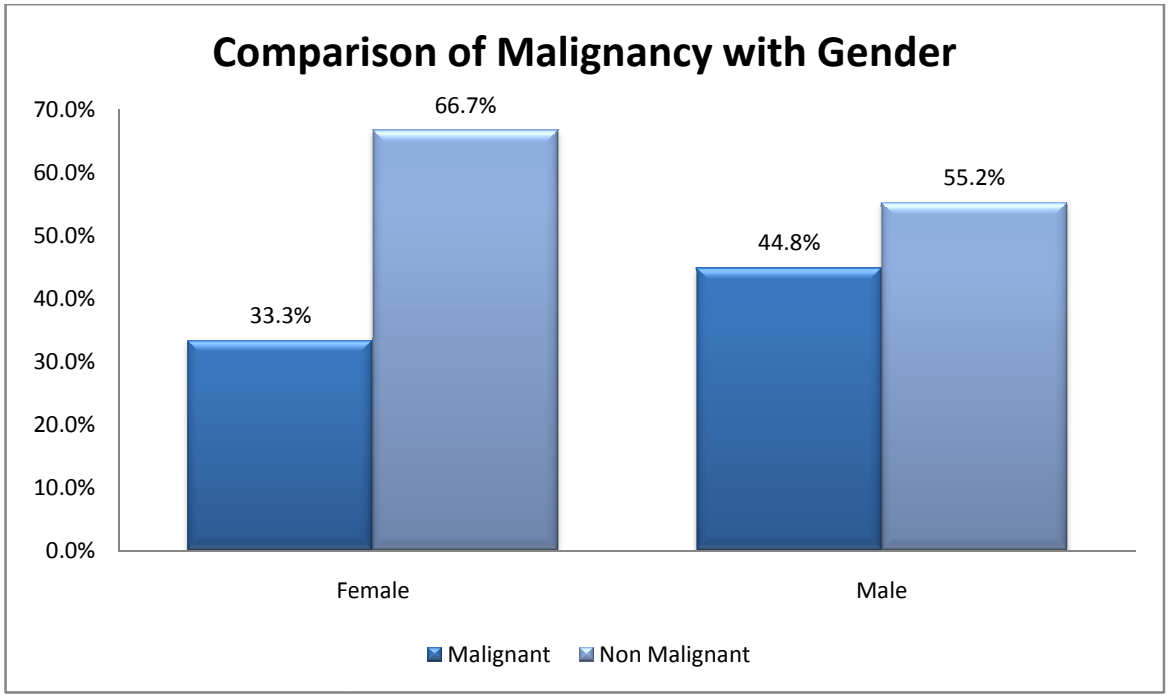


In our study of 100 cases 40 were malignant and 60 were non malignant.

DISTRIBUTION OF MALIGNANCIES BETWEEN SEX

			MNM		
			Malignant	Non Malignant	Total
SEX	FEMALE	Count	14	28	42
		% within SEX	33.3%	66.7%	100.0%
		% within MNM	35.0%	46.7%	42.0%
	MALE	Count	26	32	58
		% within SEX	44.8%	55.2%	100.0%
		% within MNM	65.0%	53.3%	58.0%
Total		Count	40	60	100
		% within SEX	40.0%	60.0%	100.0%
		% within MNM	100.0%	100.0%	100.0%

	Female	Male
Malignant	33.3%	44.8%
Non Malignant	66.7%	55.2%

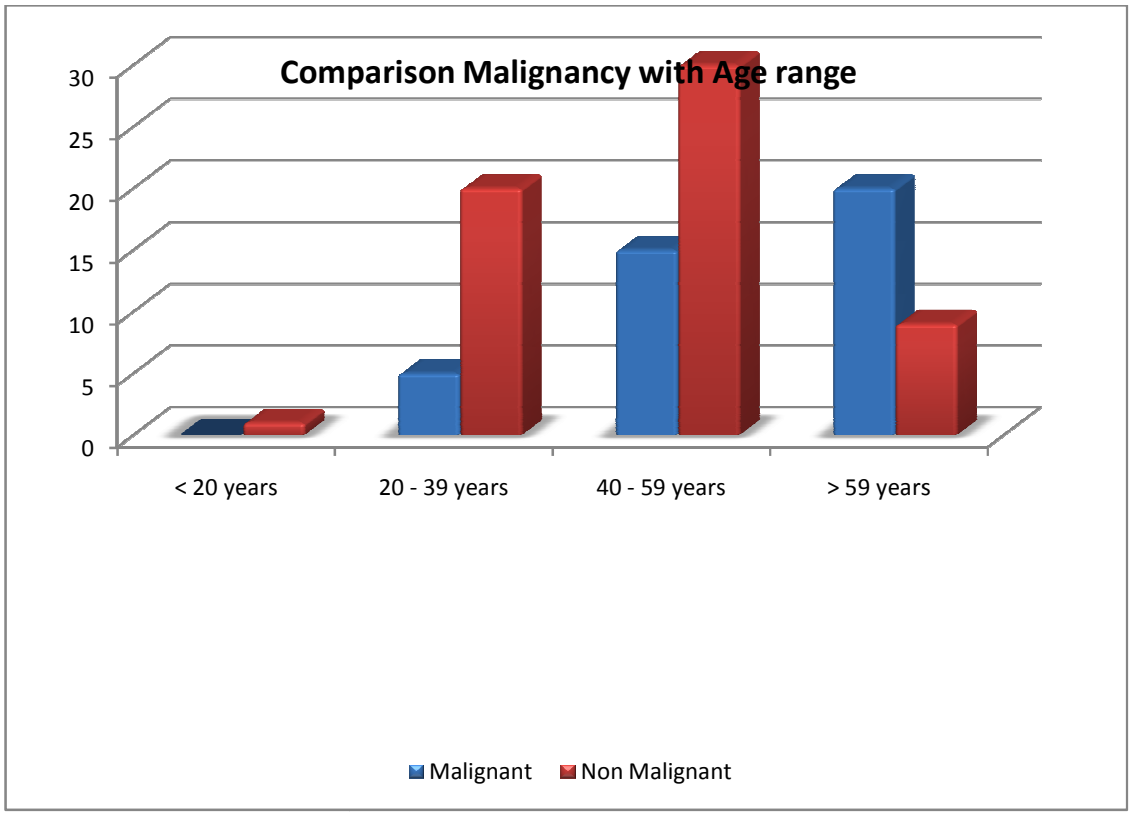


Most of the malignant subjects in our study were females (66.7%) and among the non malignant 60 subjects most of them were males (55.2 %).

DISTRIBUTION OF MALIGNANCIES AMONG VARIOUS AGE GROUPS

			MNM		
			Malignant	Non Malignant	Total
Agerange	< 20 years	Count	0	1	1
		% within AGERANGE	0.0%	100.0%	100.0%
		% within MNM	0.0%	1.7%	1.0%
	20 - 39 years	Count	5	20	25
		% within AGERANGE	20.0%	80.0%	100.0%
		% within MNM	12.5%	33.3%	25.0%
	40 - 59 years	Count	15	30	45
		% within AGERANGE	33.3%	66.7%	100.0%
		% within MNM	37.5%	50.0%	45.0%
	> 59 years	Count	20	9	29
		% within AGERANGE	69.0%	31.0%	100.0%
		% within MNM	50.0%	15.0%	29.0%
Total		Count	40	60	100
		% within AGERANGE	40.0%	60.0%	100.0%
		% within MNM	100.0%	100.0%	100.0%

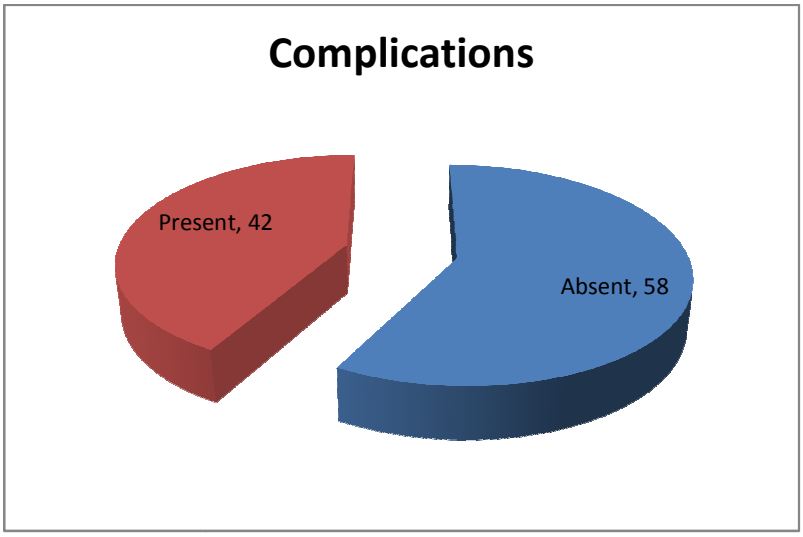
	Malignant	Non Malignant
< 20 years	0	1
20 - 39 years	5	20
40 - 59 years	15	30
> 59 years	20	9



Most of the malignant cases in our study were greater than 59 years and most of the non malignant cases were distributed in the 40 - 59 years age group.

POST SURGICAL COMPLICATIONS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	**Absent**	58	58.0	58.0	58.0
	Present	42	42.0	42.0	100.0
	Total	100	100.0	100.0	

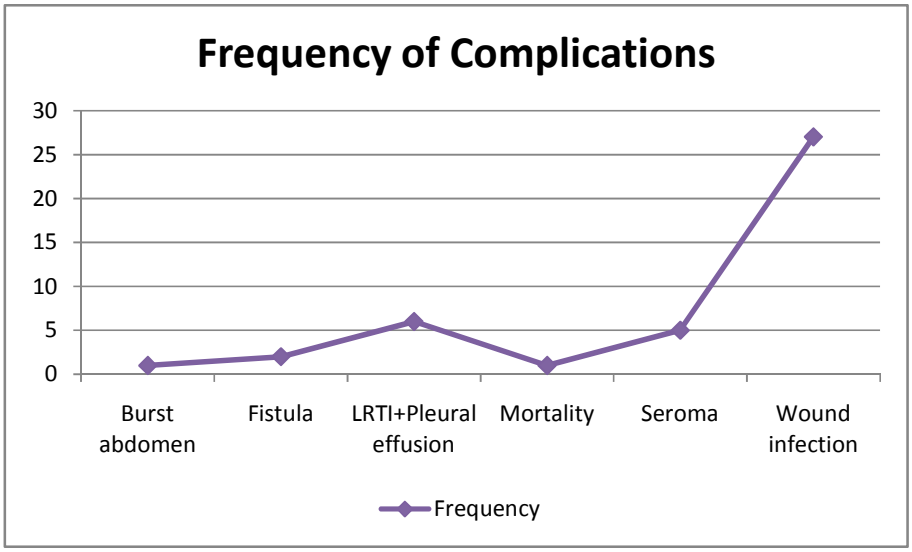


Post operative complications were present in 42 percent of the study subjects.

The varied complications and their distribution is tabulated below

COMPLICATIONS

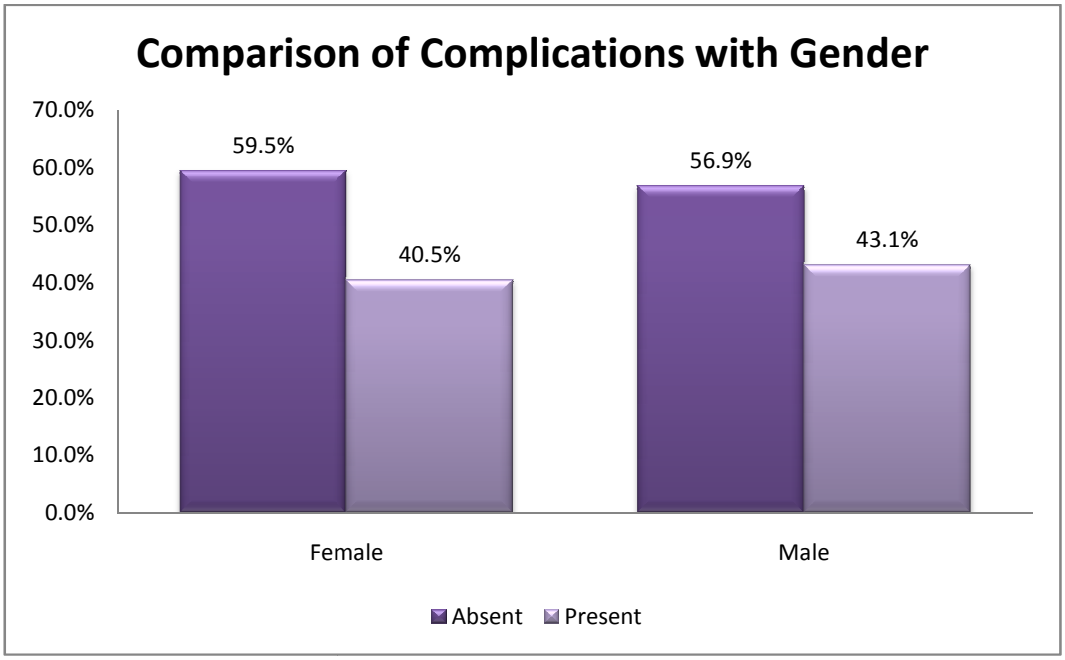
		Frequency	Percentage	Valid Percent	Cumulative Percent
Valid	Burst abdomen	1	58.0	58.0	58.0
	Fistula	2	1.0	1.0	59.0
	LRTI + Pleural effusion	6	2.0	2.0	61.0
	Mortality	1	6.0	6.0	67.0
	Seroma	5	1.0	1.0	68.0
	Wound infection	27	5.0	5.0	73.0
			27.0	27.0	100.0
	Total	100	100.0	100.0	



COMPARISON OF COMPLICATIONS WITH GENDER

			COMP		Total
			No	Yes	
SEX	**FEMALE**	Count	25	17	42
		% within SEX	59.5%	40.5%	100.0%
		% within COMP	43.1%	40.5%	42.0%
	MALE	Count	33	25	58
		% within SEX	56.9%	43.1%	100.0%
		% within COMP	56.9%	59.5%	58.0%
Total		Count	58	42	100
		% within SEX	58.0%	42.0%	100.0%
		% within COMP	100.0%	100.0%	100.0%

	Female	**Male**
Absent	59.5%	56.9%
Present	40.5%	43.1%

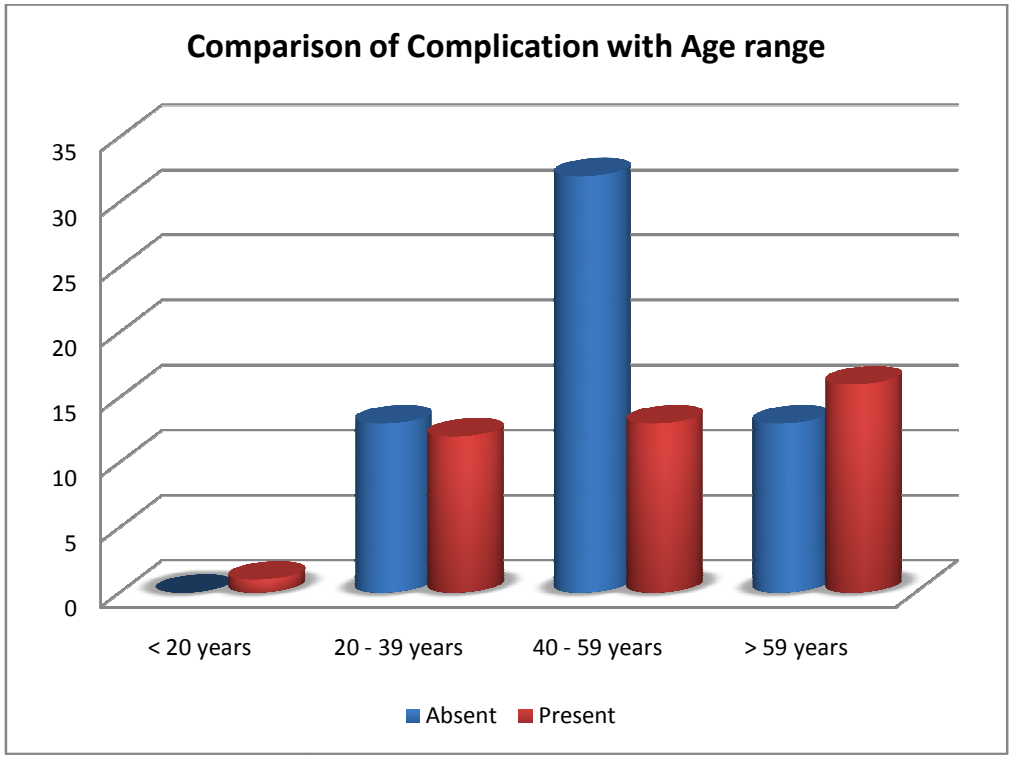


Among the female study subjects 59.5 % developed complications and among the male subjects 56.9% had morbid postoperative hospital stay with complications.

COMPARISON OF COMPLICATIONS WITH AGE RANGE

			COMP		Total
			No	Yes	
AGERANGE	< 20 years	Count	0	1	1
		% within AGERANGE	0.0%	100.0%	100.0%
		% within COMP	0.0%	2.4%	1.0%
	20 - 39 years	Count	13	12	25
		% within AGERANGE	52.0%	48.0%	100.0%
		% within COMP	22.4%	28.6%	25.0%
	40 - 59 years	Count	32	13	45
		% within AGERANGE	71.1%	28.9%	100.0%
		% within COMP	55.2%	31.0%	45.0%
	> 59 years	Count	13	16	29
		% within AGERANGE	44.8%	55.2%	100.0%
		% within COMP	22.4%	38.1%	29.0%
Total		Count	58	42	100
		% within AGERANGE	58.0%	42.0%	100.0%
		% within COMP	100.0%	100.0%	100.0%

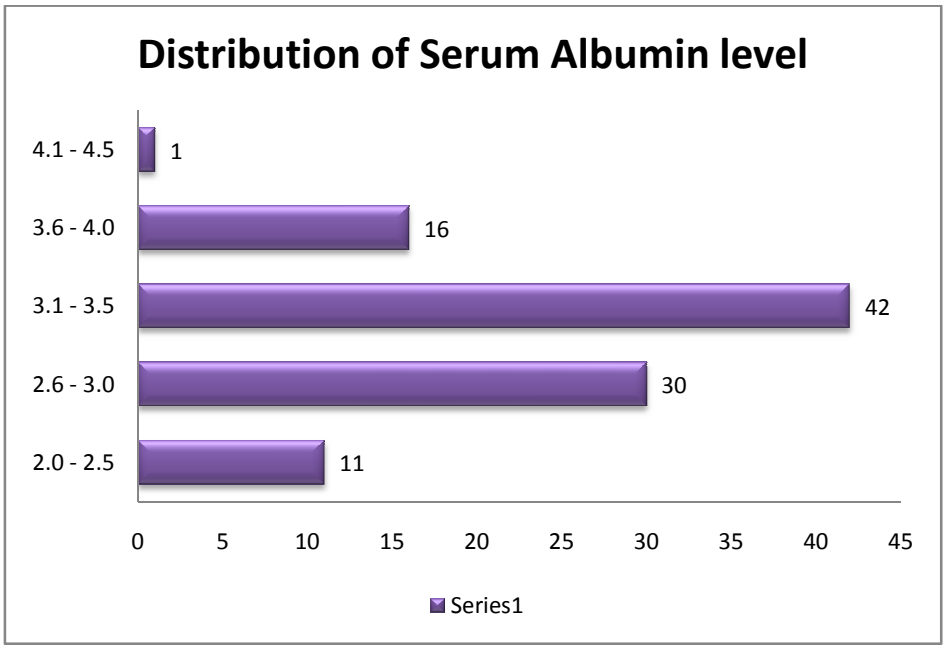
	Absent	Present
< 20 years	0	1
20 - 39 years	13	12
40 - 59 years	32	13
> 59 years	13	16



The frequency of complications increased as age advances with the maximum brunt of complications in subjects aged greater than 59years. The study subjects aged 40 - 59 years had lesser percentage of complications within their age group. In the subjects between 20 - 39 years , subjects with and without complications were equal.

DISTRIBUTION OF SERUM ALBUMIN LEVEL

SERUM AMBUMN LEVEL		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	2.0 - 2.5	11	11.0	11.0	11.0
	2.6 - 3.0	30	30.0	30.0	41.0
	3.1 - 3.5	42	42.0	42.0	83.0
	3.6 - 4.0	16	16.0	16.0	99.0
	4.1 - 4.5	1	1.0	1.0	100.0
	Total	100	100.0	100.0	

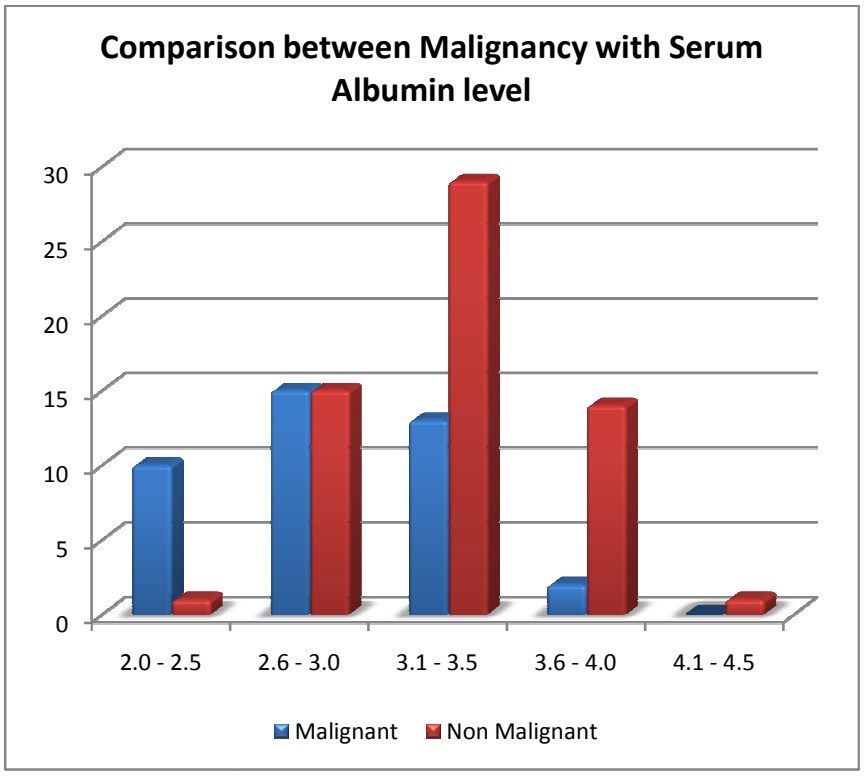


Most of the study subjects had albumin levels below desired values (less than 3.5 g/dl- 83 %) within the maximum subjects having serum albumin values between 3 and 3.5 g/dl (42%).

COMPARISON OF BETWEEN MALIGNANCY WITH SERUM ALBUMIN LEVEL

			MNM		
			Malignant	Non Malignant	Total
Serumlevel	2.0 - 2.5	Count	10	1	11
		% within SERUMLEVEL	90.9%	9.1%	100.0%
		% within MNM	25.0%	1.7%	11.0%
	2.6 - 3.0	Count	15	15	30
		% within SERUMLEVEL	50.0%	50.0%	100.0%
		% within MNM	37.5%	25.0%	30.0%
	3.1 - 3.5	Count	13	29	42
		% within SERUMLEVEL	31.0%	69.0%	100.0%
		% within MNM	32.5%	48.3%	42.0%
	3.6 - 4.0	Count	2	14	16
		% within SERUMLEVEL	12.5%	87.5%	100.0%
		% within MNM	5.0%	23.3%	16.0%
	4.1 - 4.5	Count	0	1	1
		% within SERUMLEVEL	0.0%	100.0%	100.0%
		% within MNM	0.0%	1.7%	1.0%
Total		Count	40	60	100
		% within SERUMLEVEL	40.0%	60.0%	100.0%
		% within MNM	100.0%	100.0%	100.0%

	Malignant	Non Malignant
2.0 - 2.5	10	1
2.6 - 3.0	15	15
3.1 - 3.5	13	29
3.6 - 4.0	2	14
4.1 - 4.5	0	1



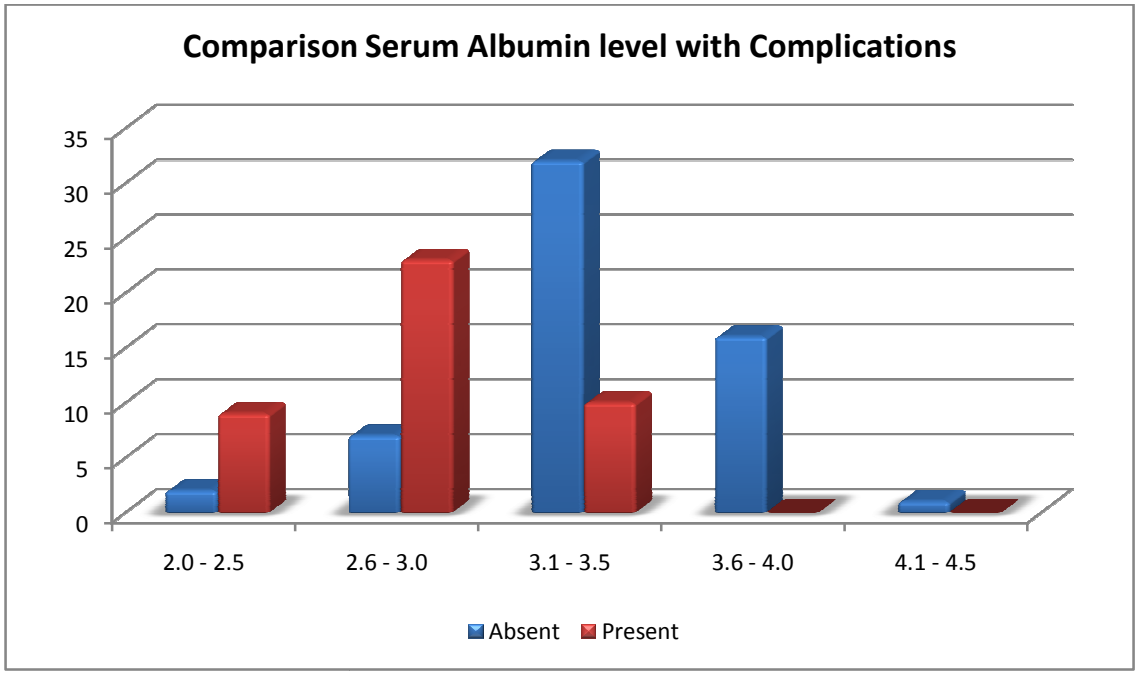
Most of the malignant cases had albumin levels less than 3 with the number of subjects maximum in the 2.6 - 3 range which was statistically significant (p value < 0.0001) Most of the non malignant cases had their albumin levels greater than 3 with the maximum number in the 3.1 to 3.5 range which was again statistically significant (p value less than 0.001)

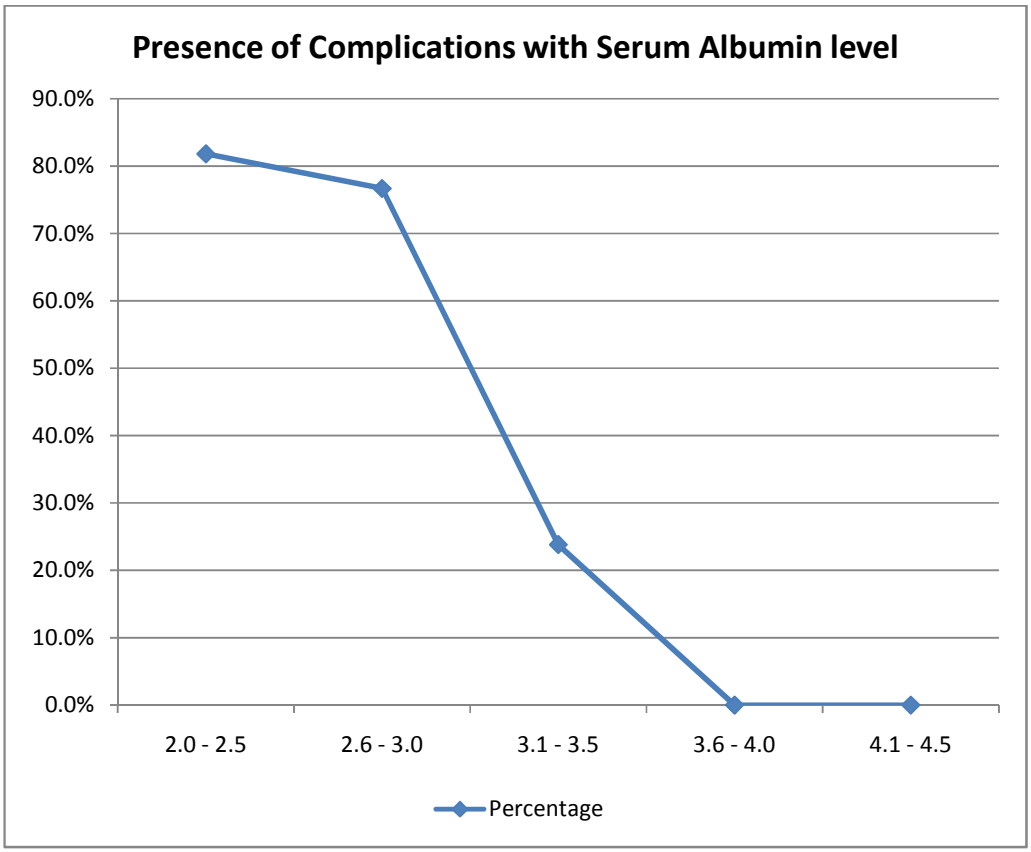
COMPARISON SERUM ALBUMIN LEVEL WITH COMPLICATIONS

The study subjects who were underwent any major abdominal surgery and developed post operative complications mostly had their albumin levels less than 3 which was statistically highly significant (p value <0.0001)

			COMP		Total
			No	Yes	
SERUMLEVEL	2.0 - 2.5	Count	2	9	11
		% within SERUMLEVEL	18.2%	81.8%	100.0%
		% within COMP	3.4%	21.4%	11.0%
	2.6 - 3.0	Count	7	23	30
		% within SERUMLEVEL	23.3%	76.7%	100.0%
		% within COMP	12.1%	54.8%	30.0%
	3.1 - 3.5	Count	32	10	42
		% within SERUMLEVEL	76.2%	23.8%	100.0%
		% within COMP	55.2%	23.8%	42.0%
	3.6 - 4.0	Count	16	0	16
		% within SERUMLEVEL	100.0%	0.0%	100.0%
		% within COMP	27.6%	0.0%	16.0%
	4.1 - 4.5	Count	1	0	1
		% within SERUMLEVEL	100.0%	0.0%	100.0%
		% within COMP	1.7%	0.0%	1.0%
Total		Count	58	42	100
		% within SERUMLEVEL	58.0%	42.0%	100.0%
		% within COMP	100.0%	100.0%	100.0%

	Absent	Present
2.0 - 2.5	2	9
2.6 - 3.0	7	23
3.1 - 3.5	32	10
3.6 - 4.0	16	0
4.1 - 4.5	1	0
	Percentage	
2.0 - 2.5	81.8%	
2.6 - 3.0	76.7%	
3.1 - 3.5	23.8%	
3.6 - 4.0	0.0%	
4.1 - 4.5	0.0%	



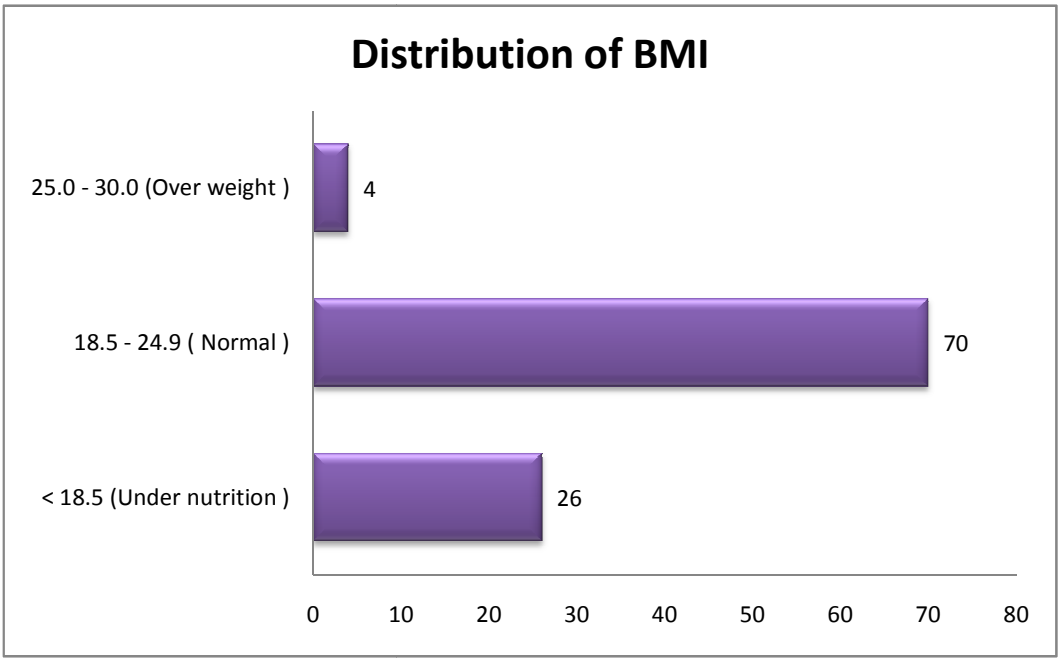


Subjects with albumin levels greater than 3.5 did not suffer any major post operative complication during hospital stay.

DISTRIBUTION OF BMI

BMI LEVEL

BMI LEVEL		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	< 18.5 (Under nutrition)	**26**	**26.0**	**26.0**	**26.0**
	18.5 - 24.9 (Normal)	**70**	**70.0**	**70.0**	**96.0**
	25.0 - 30.0 (Over weight)	**4**	**4.0**	**4.0**	**100.0**
	Total	**100**	**100.0**	**100.0**	

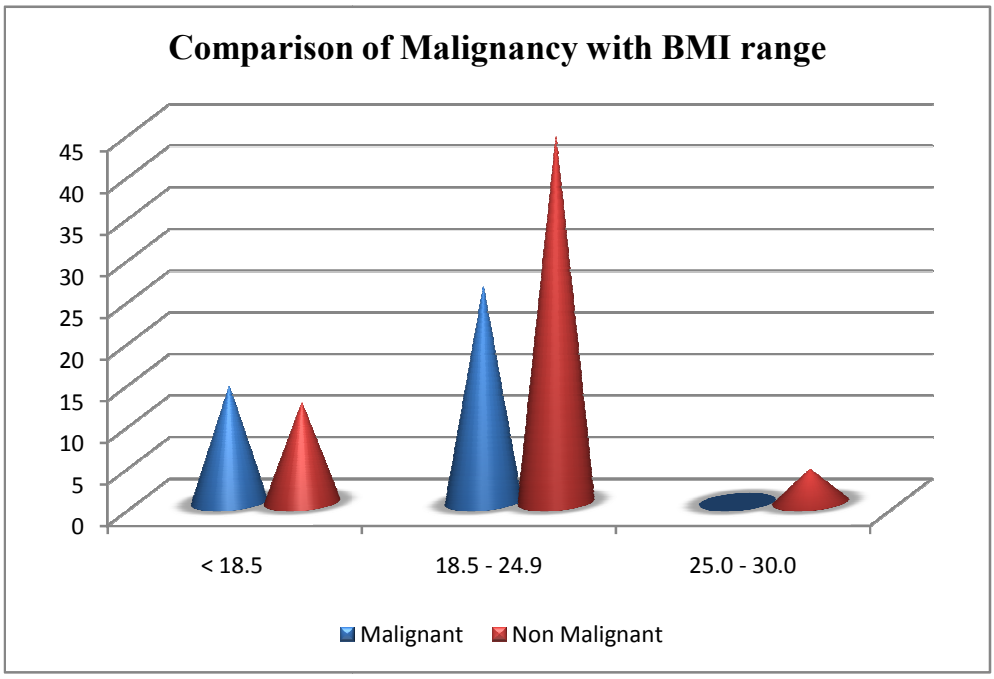


Most of the patients were distributed in the normal BMI range between 18.5 kg/m^2 and 24.9 kg/m^2. One quarter of the population under study were underweight by BMI in our study.

COMPARISON OF MALIGNANCY WITH BMI RANGE

			MNM		
			Malignant	Non Malignant	Total
BMILEVEL	< 18.5 (Under nutrition)	Count	14	12	26
		% within BMILEVEL	53.8%	46.2%	100.0%
		% within MNM	35.0%	20.0%	26.0%
	18.5 - 24.9 (Normal)	Count	26	44	70
		% within BMILEVEL	37.1%	62.9%	100.0%
		% within MNM	65.0%	73.3%	70.0%
	25.0 - 30.0 (Over weight)	Count	0	4	4
		% within BMILEVEL	0.0%	100.0%	100.0%
		% within MNM	0.0%	6.7%	4.0%
Total		Count	40	60	100
		% within BMILEVEL	40.0%	60.0%	100.0%
		% within MNM	100.0%	100.0%	100.0%

	Malignant	Non Malignant
< 18.5	14	12
18.5 - 24.9	26	44
25.0 - 30.0	0	4

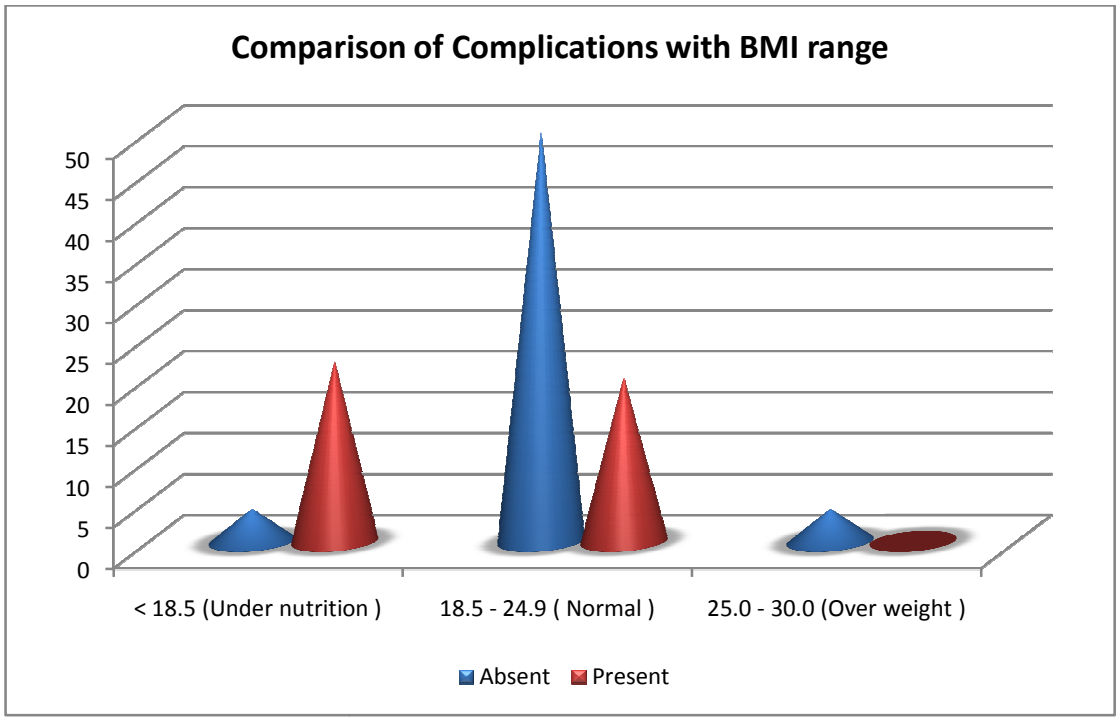


There was no significant difference statistically with regards to the distribution of BMI between the malignant and non malignant study groups.

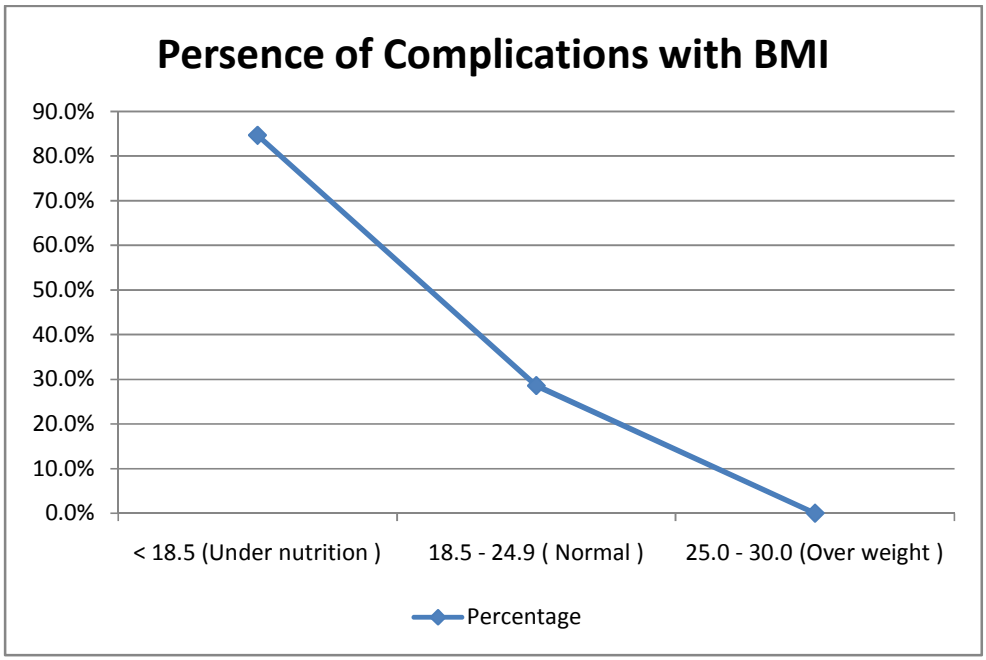
COMPARISON OF COMPLICATIONS WITH BMI RANGE

			COMP		
			No	Yes	Total
BMI LEVEL	< 18.5 (Under nutrition)	Count	4	22	26
		% within BMI LEVEL	15.4%	84.6%	100.0%
		% within COMP	6.9%	52.4%	26.0%
	18.5 - 24.9 (Normal)	Count	50	20	70
		% within BMI LEVEL	71.4%	28.6%	100.0%
		% within COMP	86.2%	47.6%	70.0%
	25.0 - 30.0 (Over weight)	Count	4	0	4
		% within BMI LEVEL	100.0%	0.0%	100.0%
		% within COMP	6.9%	0.0%	4.0%
Total		Count	58	42	100
		% within BMI LEVEL	58.0%	42.0%	100.0%
		% within COMP	100.0%	100.0%	100.0%

	Absent	Present
< 18.5 (Under nutrition)	4	22
18.5 - 24.9 (Normal)	50	20
25.0 - 30.0 (Over weight)	4	0

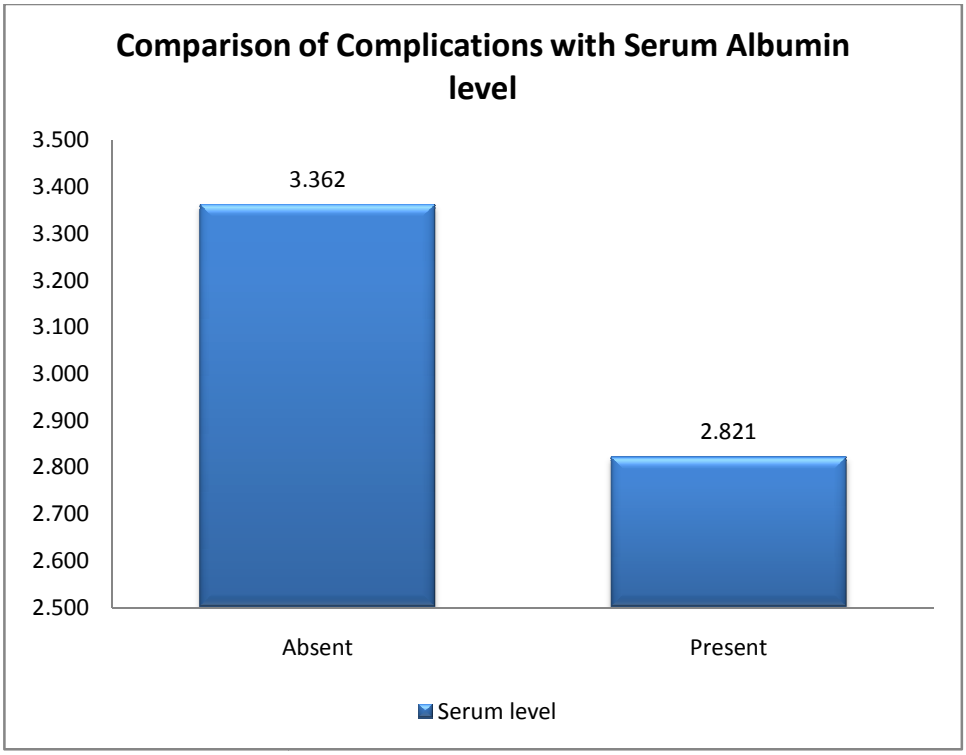


	Percentage
< 18.5 (Under nutrition)	84.6%
18.5 - 24.9 (Normal)	28.6%
25.0 - 30.0 (Over weight)	0.0%



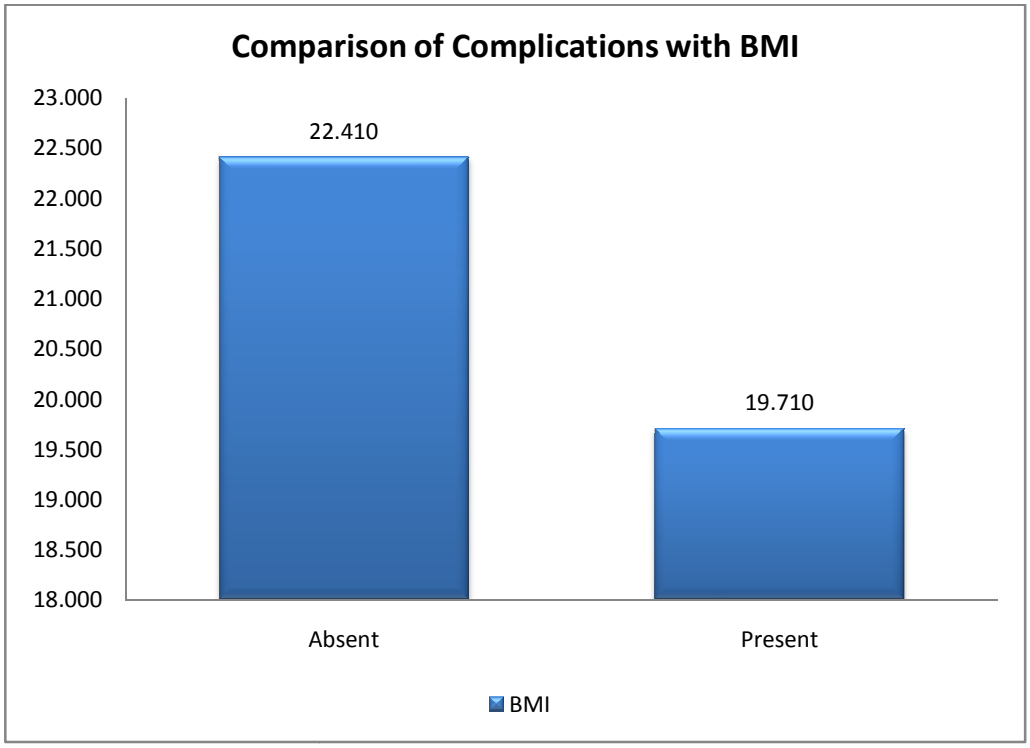
There was a statistically significant increase in the number of complications as BMI decreases with the maximum percentage of complications in the underweight BMI group.

MEAN SERUM ALBUMIN AND BMI WITH REGARDS TO INCIDENCE OF COMPLICATIONS



On an average patients with albumin levels averaging around 2.8 g/dl preoperatively were subjected to post operative complications in our observation.

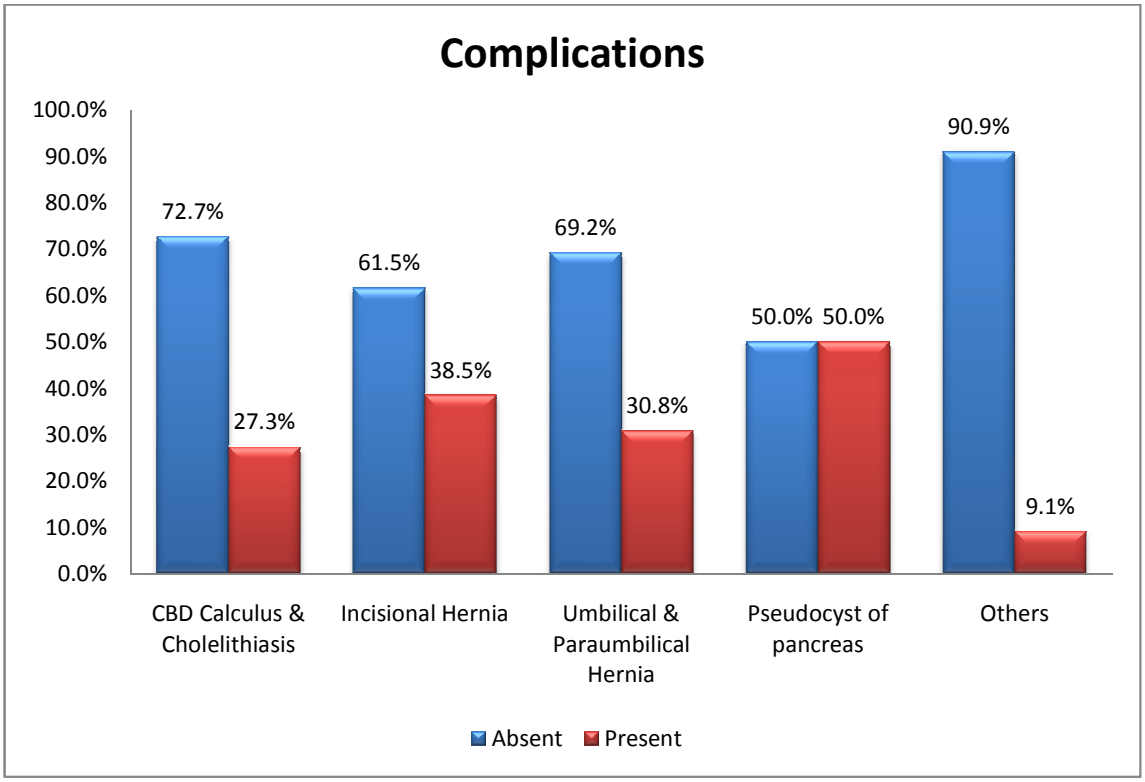
And further patients with 3.3 g/dl and higher had lesser incidence of complications postoperatively.



BMI around 19.2kg/m^2 as the mean was associated with higher postoperative complications and BMI of 22.4 kg/m^2 and higher was associated with lesser complication rates.

COMPLICATIONS IN NON MALIGNANT CASES

			COMPLICATIONS		
			Absent	Present	Total
DIAGNOSIS	CBD Calculus & Cholelithiasis	Count	8	3	11
		% within DIAGNOSIS	72.7%	27.3%	100.0%
	Incisional Hernia	Count	8	5	13
		% within DIAGNOSIS	61.5%	38.5%	100.0%
	Umbilical & Paraumbilical Hernia	Count	9	4	13
		% within DIAGNOSIS	69.2%	30.8%	100.0%
	Pseudocyst of pancreas	Count	6	6	12
		% within DIAGNOSIS	50.0%	50.0%	100.0%
	Others	Count	10	1	11
		% within DIAGNOSIS	90.9%	9.1%	100.0%
Total		Count	41	19	60
		% within DIAGNOSIS	68.3%	31.7%	100.0%



With special regards to non malignant cases, the incidence of post operative complications was maximum with a preoperative diagnosis of pseudocyst of pancreas with patients in the group having serum albumin 3.2g/dl on an average and 21 kg/m^2 as BMI on an average, undergoing cystogastrostomy followed by open incisional hernia repair undergoing hernioplasty.

DISCUSSION

Organic defence decrease and malnutrition were recognised as potential factors for higher morbidity and mortality rates in the post operative period. Malnourished patients are at a higher risk of postoperative complications and death, if compared to well nourished patients subjected to similar surgeries.

Nutritional assesment is therefore essential in order to identify patients who are at an increased risk of developing post operative complications. Though numerous studies have evaluated different nutritional indices , serum albumin and body mass index as markers of assesing post operative morbidity and mortality have been taken up in certain studies.

Serum albumin levels less than 3g/dl was associated with increased post operative morbidity according to the study by Mullen et al, Golub et al , Leite et al.

Our study also attributes itself to similar results with patients having albumin levels lesser than 3 g/dl suffering from significantly higher complications in the form of seroma , wound gaping, lower respiratory tract infections, mortality , fistula when compared with patients having higher albumin levels. Most of the patients in our study had albumin levels ranging from 3.1 to 3.5 g/dl

Gibbs et al observed in his study that a fall in serum albumin levels from 4.6 g/dl to 2.1 g/dl was associated with exponential increase in mortality rates from less than 1% to 29% in morbidity rates from 10% to 65%. Albumin thus was evaluated as the strongest predictor of mortality and morbidity for surgery as a whole. It was a better predictor of some types of morbidity, particularly sepsis and major infections , than other types and was a very useful prognostic marker according to the study.

The post operative complications were more in female than males in the above studies. Our study also had a slight preponderence to females with respect to complications during post operative hospital stay.

Complications also had an exponential increase as age advances as is also evident in our study with complications more with age groups greater than 59 years .

Varut Lohsiriwat et al in their study demonstrated that pre operative hypoalbunemia is a major risk factor for post operative complications following rectal cancer surgeries. This suggests pre operative hypoalbuminemia is an independent risk factor for post operative complications following rectal cancer surgeries as well as post operative bowel function and hospital stay. Our study also shows that lower serum albumin levels and increased incidence of complications among the malignat population as compared to the non malignant patients.

Table 11: Significance of serum albumin levels in predicting postoperative outcomes.

Study name	Sr. Alb g/dl associated with increased complications	P value
Beghetto et al	<3.5	<0.05
Leite et al	<3	<0.05
Brown et al	<3	<0.05
Engelman et al	<2.5	<0.001
Foley et al	<2.5	<0.001
Present study	<3	<0.001

Engelman et al observed that albumin levels less than 2.5 g/dl and BMI less than 20 kg/m2 was associated with increased post operative complications.

In our study the post operative complications increased as the albumin levels decreased from 3 g/ dl to lesser levels and also as regards BMI, the complications were more with patients with underweight measuring less than 18.5 kg/m^2.

Similar results were also put forward by a prospective cohort study done by Mullen et al on impact of Body Mass Index on perioperative outcomes in patients undergoing major intra abdominal surgeries where they concluded

that patients who were underweight had a fivefold increase in post operative mortality , perhaps as a consequence of their underlying nutritional status.

In a study by Michael et al on malnutrition , outcome and nutritional support suggest pre operative nutritional risk indications like BMI < 18.5 kg/m^2 and serum albumin < 2.1 g/dl have an impact on surgical outcome. Such patients are evidently malnourished and consequently have longer hospital stays and experience a 40 -60% greater frequency of complications in response to medical / surgical treatment.

In addition our study also evaluated the differences in complications among the non malignant cases and revealed that post operative complications were at a higher incidence among patients with a preoperative diagnosis of pseudocyst of pancreas undergoing cystogastrostomy followed by incisional hernia undergoing hernioplasty.

CONCLUSION

Serum albumin is thus validated as a single important marker indicative of post operative complications , depicting the individual's nutritional status and highly indicative of post operative morbidity and mortality.

In our study of 100 patients, the complication rate was more when serum albumin level was less than 3 g/dl which was statistically significant. The rate of complications decreased as serum albumin level increased from 3.1 g/dl and above. Patients with serum albumin level greater than 3.5 g/dl had lesser complications which was statistically significant irrespective of malignant or non malignant disease pathology.

Thus serum albumin is a better predictor of surgical outcomes than many other preoperative patient characteristics. It is comparabily a low cost test that is widely performed and with its esteemed value should be used more frequently as a prognostic tool to detect malnutrition and risk of adverse surgical outcomes.

BMI in underweight category is also associated with statistically significant post operative complications.

There was no statistically significant sex preponderence with regards to complication rates.

The disease process also plays a role in post operative complication rates as evidenced in our study as the complication rates were higher in the malignant population which was statistically significant and among the non malignant group, complications were more with patients having pseudocyst of pancreas undergoing cystogastrostomy.

Thus serum albumin and BMI can be used as an easy, cost effective and highly reliable tools in predicting post operative complications and thus effective preoperative nutritional supplementation can be given to reduce morbidity and mortality in the long run.

BIBLIOGRAPHY

1. John MacFie; Nutrition and Fluid Therapy. Bailey and Love, Short Practice of Surgery 25th edn, 223.
2. N.Engl. J. Med 1991; 325:52; The Washington Manual of Surgery;5th edition
3. N. Engl. J. Med 1991; 325:525 Arch surg 1999; 134:36; The Washington Manual of Surgery; 5th edition.
4. Robert B Baron Nutrition – Assessment of nutritional status. Current Medical Diagnosis and Treatment 2006;1263
5. Badac V. Jan, Stephen F. Lowry Systemic response to injury and metabolic support; 9th edition, 40 Shwartz Principles of Surgery.
6. AdrionBarbul, David T Efron Wound Healing; Shwartz principles of Surgery; 9th edition, 210
7. Sean C. Glasgow, Virginia M. Hermann, Surgical metabolism and nutrition. Current Surgical Diagnosis and Treatment, 12th edition, 140-44
8. Hugo Q. Cheng preoperative evaluation and perioperative management. Current Medical Diagnosis and Treatment 2010; 53-54.
9. Anne Coble Voss, Kathleen E. Mayer. Role of liquid dietary supplements. Nutrition in the prevention and treatment of disease;465-66.

10. Allison, D.B., Gallagher,D., Heo.M., et al. BMI and all cause mortality any people age 70 and over: The longitudinal study of aging. Int.J.Obes 21, 424-431.
11. Bonnie A. Spear. Nutrition in adolescence. Krause's Food, Nutrition and Diet therapy; 11th edition, 286,330 Robbins, Cotrans. Tissue Renewal and Repair. Regeneration, Healing and Fibrosis. Robbins and Cotran Pathologic Basis of Diseases; 7thedition; 107
12. Dorothy B.Doughty, Bonnie Sparks-Defriese Wound Healing Physiology, Acute and Chronic of Modern Wounds Current Management Concepts; 3rd edition; 57
13. Mahmood N. K, Marril T.D, Surgical Complications, Sabiston Textbook of Surgery, The biologic basis of Modern Surgical Practice; 18th edition; 337
14. Margaret L.R, Robert K.M. Plasma proteins, Immunoglobulins and Blood Coagulation. Harpers Biochemistry; 25th edition, 740
15. McPhee BJ, Lingappa VR. Circulation –Circulating body fluids, Ganong Review of Medical Physiology; 22nd edition,540
16. Ian A.D. Boucher, Harold Ellis, Peter L. Fleming. Albumin Foerch's Index of Differential diagnosis; 13th edition, 735
17. Gibbs J, Cull W, Henderson W. Daley J, Hur K, Khuri SF. Preoperative serum albumin level as a predictor of operative mortality and morbidity. Arch Surg 1999;134:36-42

18. Kudsk KA, Tolley EA, Delvitt RC, Janu PG, Blackwell AP, Kin BK et al. Preoperative albumin and surgical site identify surgical risk for major postoperative complications. JPEN J Parenter Enteral Nutr 2003; 27(1):19

19. Palma S, Cosano A, Meriscal M, Martinez – Gallago G, Medina – Cuadros M, Delgado – Bodriguez M. Cholesterol and Serum albumin as risk factors for death in patients undergoing general surgery. Br. J Surg 2007; 94:369-375

20. M.B. BadiaTahulla, J. Llop-Talaverona, R. JadarMasanesa. Preoperative albumin as a predictor of outcome in Gastrointestinal surgery online, Vol 4 Issue 5 2009; 248-51

21. Detsky A S, Bakee J P, O Rourke. Predicting nutrition associated complications for patients undergoing gastrointestinal surgery. JPEN J Parenter Enter Nutr 1987; 11:440-446

22. Vincent JL, Dubois MJ, Navicks RJ, Wilkes MM. Hypoalbuminemia in acute illness: Is there a rationale for intervention? Ann Surg 2003; 237:319-34

23. Foley EF, Borlase BL, Dzik WH. Albumin supplementation in the critically ill: A prospective, randomized trial. Arch Surg 1990;125:739-42

24. Woods MS, Kelly H. Oncotic pressure, albumin and ileus: The effect of albumin replacement on post operative ileus. Ann Surg 1993 Nov; 59(11):758-63

25. Brown RO, Bradley JE, Bekemey WB. Effect of albumin supplementation during parenteral nutrition on hospital morbidity. Crit care Med 1988;16:1177-1182

26. Golub R, Sorrento JJ Jr, Cantu R Jr. Efficacy of albumin supplementation in the surgical intensive care unit: A prospective, randomized study. Crit Care Med 1997; 25:249-52

27. MG Beghetto, VC Luft, ED Mello. Accuracy of nutritional assessment tools for predicting adverse hospital outcomes. Nutr Hosp. 2009; 24 (1):56-62

28. Liop JM, Munoz C, Badia MB, Virgili N,Tubau M, Ramon JM et al. Serum Albumin as an Indicator Clinical Evaluation in Patients on Parenteral Nutrition. Multivariate study. ClinNutr 2001; 20(1): 77-81.

29. Anderson CF, Moxness K, Meister J, Burritt MF. The Sensitivity and Specificity of nutrition-related variables in relationship to the duration of hospital stay and the rateof complications. Mayo ClinProc 1984; 59(7): 477-83.

30. Hirsh S, De Obaldia N, Petermann M, Coracevice S, Burmerstee R, Lorm P. Nutritional status of surgical patients and the relationship of nutrition to postoperative outcome. J Am CollNutr 1992; 11(1): 23- 24

31. Leite HP, Fisberg M, De Carvallio WB. Serum albumin and clinical outcomes in paediatric cardiac surgery. Nutrition 2005; 21(5):553-58
32. EngelmanDt, Adams DH, Byrne JG,Avanki SF, Collins JJ, Coupee GS et al. Impact of BMI and serum albumin on morbidity and mortality after cardiac surgery. J ThoracCardiovasSurg 1999; 118:866-73
33. Mullen JJ, Davenport DL, Hutter MM, Hosokawa PW, Henderson WG, KhuriSf et al. Impact of BMI on perioperative outcome in patients undergoing major interabdominal cancer surgery. Ann SurgOnco 2008; 15:2164-72
34. Arozullah A, Daley J, Henderson W et al Multifactorial risk index for predicting postoperative respiration failure in men after non cardiac surgery. Ann Surg 2000; 232: 243-53
35. Daniel L Davenport, Victor A Ferraris, Patrick Hosokawa, William G Henderson, Shukri K Khuri, SRobert M Mentzer. Multivariable predictors of post operative adverse events after General and Vascular Surgery: Results from Patient Safety in Surgery Study
36. VarutLohsiriwat, Darin Lohsiriwat, WiroAkaraviputti et al. PreoponBoonnoch, ThawatchaiAkaraviputti et al. Preoperative hypoalbuminemia is a major risk factor for post operative complication following rectal cancer surgery. World Journal Gastroentero 2008 Feb, 28; 14(8): 1248-51

37. Azodi SA, Lindstorm D, Adami J, BellocoR, Linder S, Wladis A. Impact of body mass index and tobacco smoking on outcome after open appendicectomy. Br. J. Surg 2008; 95:751-57 38 Jennifer L. Gnerlich and Timothy G. Buchman; Fluid, Electrolyte, and Acid-Base Disorders. The Washington Manual of Surgery, 5thedn, 85.

PROFORMA

NAME: D.O.A:

AGE: D.O.D:

SEX: IP/OP NO:

EDUCATION:

OCCUPATION:

ADDRESS:

CHIEF COMPLAINTS:

HOPI:

PAST HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

GENERAL PHYSICAL EXAMINATION:

HEIGHT_____cms

WEIGHT_____kgs

BMI: <18.5

18.5-24.9

25-29.9

>30

PULSE_____/min

BP_____/_____mm/hg

PALLOR:

ICTERUS:

PEDAL EDEMA:

LYMPHADENOPATHY:

SYSTEMIC EXAMINATION:

RS:

CVS:

PA:

LOCAL EXAMINATION:

DIAGNOSIS:

INVESTIGATIONS:

BLOOD ROUTINE:

URINE ROUTINE:

SERUM ALBUMIN:

<3.5g/dl

>3.5g/dl

MANAGEMENT:

SURGERY PERFORMED:

POSTOPERATIVE PROGRESS:

WOUND INFECTION :

DISCHARGE :

LEAK :

DEHISCENCE :

OTHERS:

MORTALITY:

MASTER CHART

S. No	NAME	AGE/SEX	IP.NO	DIAGNOSIS	TREATMENT	MALIGNANT/NON-MALIGNANT	SERUM ALBUMI	BMI	COMPLICATIONS-	COMPLICATIONS
1	Aashiya Begum	44/F	36783	Retroperitoneal GIST	Wide local excision with splenectomy with distal pancreatectomy with partial gastrectomy	MALIGNANT	2.7	18.1	yes	wound infection
2	Achikam	60/F	28872	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.9	19.8	yes	wound infection
3	Ali Rehmath	53/F	7705	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	3.4	22.8	yes	seroma
4	Ammaiyappan	64/M	21731	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	3.3	19.5	yes	wound infection
5	Appavu	72/M	45254	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.4	17.9	yes	LRTI+pleural effusion
6	Arun Prakash	35/M	75913	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.8	26.1	no	
7	Ashwin	15/M	10176	Ventral Hernia	Open Hernioplasty	NON-MALIGNANT	2.9	18.3	yes	wound infection
8	Balan	30/M	16825	Retroperitoneal Mass	Excision	MALIGNANT	3.2	20.5	no	
9	Balasubramani	52/M	62626	Pseudocyst of pancreas	Cystgastrojejunostomy	NON-MALIGNANT	3.1	23.8	yes	wound infection
10	Barkhath Ali Khan	30/M	29750	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	3.9	23.5	no	
11	Chandrashekar	60/M	28874	Periampullary Carcinoma	Whipple's Procedure	MALIGNANT	2.4	18	yes	fistula
12	Chelladurai	76/M	75709	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.9	17.7	yes	LRTI+pleural effusion
13	Chinnal	65/F	10682	Carcinoma stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.9	20.8	yes	wound infection
14	Deepa	31/F	4686	Incisional Hernia	Open hernioplasty	NON-MALIGNANT	3.1	24	yes	seroma
15	Deivanai	52/F	14352	Splenic Flexure Growth	Left Hemicolectomy	MALIGNANT	3.2	20.2	no	
16	Devaraj	40/M	71623	Cholelithiasis	Open Cholecystectomy	NON-MALIGNANT	4	23.7	no	
17	Dhandapani	46/M	4642	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.3	18.4	no	
18	Durgadevi	20/F	50879	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.3	17.7	yes	wound infection
19	Ganesan	60/M	50392	Carcinoma Esophagus	Transhiatal esophagectomy	MALIGNANT	2.8	20.5	yes	wound infection
20	Gopal	67/M	53321	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.9	22.1	no	
21	Govindasamy	56/M	74152	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	3.3	23.4	no	
22	Ishwari	45/F	19704	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	3.8	20.3	no	
23	Janaki	44/F	4220	Subacute appendicitis	Open appendicectomy (others)	NON-MALIGNANT	4.4	23.2	no	
24	Jebamalraj	56/M	71453	Subacute intestinal obstruction with stricture ileum	Resection with ileotransverse anastomosis (others)	NON-MALIGNANT	3.2	23.8	no	
25	Jemini	57/M	31193	Periampullary Carcinoma	Whipple's Procedure	MALIGNANT	2.8	18.8	yes	mortality

26	Jennith Nisha	43/F	3064	Subacute cholecystitis	Open cholecystectomy (others)	NON-MALIGNANT	3.5	24.8	no	
27	Jeyaraj	32/M	16854	Carcinoma Rectum	APR with End Colostomy	MALIGNANT	2.8	20.2	yes	wound infection
28	Jothimani	43/F	21773	Adenocarcinoma Gallbladder	Open Cholecystectomy	MALIGNANT	2.3	17.8	yes	wound infection
29	Kaliveeran	77/M	48847	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.5	18.3	yes	wound infection
30	Kaliyammal	48/F	46611	Cholelithiasis	Open Cholecystectomy	NON-MALIGNANT	2.9	17.9	yes	wound infection
31	Kalleshwari	30/F	59548	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	3.7	25.8	no	
32	Kannamal	47/F	26230	Choledocholithiasis	Cholecystectomy with CBD exploration	NON-MALIGNANT	3.1	24.1	no	
33	Kannan	66/M	163446	Splenic Flexure Growth	Left Hemicolectomy	MALIGNANT	2.9	18.3	yes	wound infection
34	Kannan	36/M	76509	TAO left lower limb	Lumbar sympathectomy (others)	NON-MALIGNANT	3.9	23.4	no	
35	Karuppasamy	75/M	78968	Rectosigmoid growth	APR with End Colostomy	MALIGNANT	2.9	21.5	yes	burst abdomen
36	Karuppasamy	31/M	76800	Cholelithiasis	Open Cholecystectomy	NON-MALIGNANT	2.8	17.8	yes	wound infection
37	Kavitha	34/M	62539	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	2.9	18.4	yes	wound infection
38	Krishnan	67/M	3842	CA ANAL CANAL	APR with End Colostomy	MALIGNANT	3.2	18.7	no	
39	Krishnan	75/M	26221	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2	18.4	yes	LRTI+pleural effusion
40	Kumar	34/M	11614	Rectosigmoid growth	APR with End Colostomy	MALIGNANT	3.2	20.4	no	
41	Lakshmi	40/F	42213	Calculous cholecystitis	Open Cholecystectomy	NON-MALIGNANT	3.5	22.8	yes	wound infection
42	Lakshmi	35/F	3365	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	2.7	18	yes	wound infection
43	Lakshmi	63/F	36297	Paraumbilical Hernia	Open Hernioplasty	NON-MALIGNANT	3.5	24	no	
44	Lathadevi	45/F	13739	Carcinoma stomach with liver secondaries	Palliative Anterior Gastrojejunostomy	MALIGNANT	3	20.3	no	
45	Madan	34/M	19165	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.5	24	no	
46	Madari	30/F	30868	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	2.9	23.2	yes	seroma
47	Maheswari	38/F	53372	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	4	25.2	no	
48	Mani	61/M	32274	Incisional Hernia	Hernioplasty	NON-MALIGNANT	2.9	18.4	no	
49	Manikandan	48/M	43530	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.4	22.1	yes	LRTI+pleural effusion
50	Manjula	45/F	29703	Carcinoma Rectum	Low Anterior Resection with colostomy	MALIGNANT	3.2	21.2	no	

51	Mariappann	36/M	61516	Pseudocyst of pancreas	Cystgastrojejunostomy	NON-MALIGNANT	2.7	22.8	yes	LRTI+pleural effusion
52	Mariyayee	37/F	69022	Carcinoma Head of pancreas	Palliative Triple bypass	MALIGNANT	2.3	18	yes	wound infection
53	Michel	46/M	50989	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	3.7	22.7	no	
54	Murali	25/M	29770	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	2.8	19.1	yes	fistula
55	Murugan	48/M	70946	Periampullary Carcinoma	Whipple's Procedure	MALIGNANT	3.4	21.1	no	
56	Murugan	40/M	7765	Choledocholithiasis	CBD exploration	NON-MALIGNANT	3.7	23.2	no	
57	Murugan	47/M	68410	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	2.7	18.1	no	
58	Murugesan	50/M	42325	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.9	21	yes	wound infection
59	Myilsamy	75/M	79423	Gastrointestinal Stromal Tumour	Excision	MALIGNANT	2.9	20	no	
60	Nagarathinam	52/F	17013	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	3.5	23.5	no	
61	Nataraj	63/M	30679	Tuberculous Abdomen with ileal stricture	Resection with ileoclic anastomosis (others)	NON-MALIGNANT	2.4	18.2	no	
62	Omana	49/F	13079	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	4	22.4	no	
63	Palanisamy	60/M	31182	Synchronous Adenocarcinoma Rectum and Descending colon	Hartman s procedure with end colostomy	MALIGNANT	2.8	18.3	yes	wound infection
64	Palanisamy	37/M	58375	Carcinoma Sigmoid Colon	AR with end colostomy	MALIGNANT	3.2	21	yes	wound infection
65	Pappathy	47/F	33124	Carcinoma Rectum	APR with End Colostomy	MALIGNANT	3.2	22.5	no	
66	Pongiammal	63/F	43043	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	3.4	21.1	no	
67	Ponnammal	60/F	4530	CBD calculus	Cholecystectomy with CBD exploration	NON-MALIGNANT	3.3	23	no	
68	Ponnuram	68/M	8467	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	3.3	21.8	no	
69	Poongodi	85/F	3074	Subacute appendicitis	Open appendicectomy (others)	NON-MALIGNANT	3.8	24.5	no	
70	Poovathal	45/F	30892	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	2.6	18.1	yes	seroma
71	Rajalakshmi	45/F	3066	Incisional Hernia	Open hernioplasty	NON-MALIGNANT	3.8	24.9	no	
72	Rajamani	62/F	38291	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	2.8	17.9	yes	wound infection
73	Rajammal	80/F	36241	Cholelithiasis	Open Cholecystectomy	NON-MALIGNANT	3.1	23	no	
74	Rajan	50/M	30952	Carcinoma Anal Canal	End Colostomy	MALIGNANT	3.3	20.1	no	
75	Rajathangam	53/F	38691	Paraumbilical Hernia	Open Hernioplasty	NON-MALIGNANT	3.1	22.7	no	

76	Rajeswari	32/F	47786	Calculous cholecystitis	Cholecystectomy with CBD exploration	NON-MALIGNANT	3.3	24.2	no	
77	Rajeswari	30/F	18445	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	3.2	22.8	no	
78	Ramai	75/F	45798	Carcinoma Rectum	APR with End Colostomy	MALIGNANT	2.4	18.1	yes	LRTI+pleural effusion
79	Ramajayam	61/M	40483	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	2.7	23.2	no	
80	Ramalakshmi	47/F	42596	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	3.4	22.8	no	
81	Raman	39/F	3330	Subacute appendicitis	Open appendicectomy (others)	NON-MALIGNANT	3.4	23.7	no	
82	Ramasamy	57/M	51370	Chronic calcific	Puestow s procedure (others)	NON-MALIGNANT	3.1	21.8	no	
83	Ramaswamy	58/M	10825	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.4	23.8	no	
84	Ranganath	55/M	41817	Incisional Hernia	Open Hernioplasty	NON-MALIGNANT	3.6	26.3	no	
85	Rangasamy	49/M	40145	Umbilical hernia	Open Hernioplasty	NON-MALIGNANT	3.4	22.5	no	
86	Ravi	43/M	56263	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.7	21.2	no	
87	Sangeetha	23/F	32245	Cholelithiasis	Open Cholecystectomy	NON-MALIGNANT	3.4	22.8	no	
88	Sapan Misthai	40/M	59587	Subacute appendicitis	Open appendicectomy (others)	NON-MALIGNANT	3.8	24.2	no	
89	Sebaraj	56/M	28559	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.4	22.2	yes	wound infection
90	ShalathMani	55/M	1337	Hydatid cyst liver	Excision with marsupilisation (others)	NON-MALIGNANT	3.2	23.4	no	
91	Shanmugam	69/M	31394	Acute cholecystitis	Open Cholecystectomy	NON-MALIGNANT	3.3	23.2	no	
92	Shivalingam	57/M	29614	Carcinoma Rectosigmoid junction	Anterior Resection	MALIGNANT	2.1	18.2	yes	wound infection
93	Suburaj	56/M	28559	Chronic calcific	Puestow s procedure (others)	NON-MALIGNANT	2.7	17.8	yes	seroma
94	Sundarambal	67/F	5669	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	2.8	21.4	yes	wound infection
95	Sunderraj	56/M	30742	Carcinoma Stomach	Billroth II Gasterectomy with Jejunojejenostomy	MALIGNANT	3.4	20.1	no	
96	Thambidurai	56/M	5629	Pseudocyst of pancreas	Cystogastrostomy	NON-MALIGNANT	3.1	22.7	no	
97	Vetrivel	38/M	28943	Paraumbilical Hernia	Open Hernioplasty	NON-MALIGNANT	2.7	20.7	no	
98	Vijaya	30/F	73471	Paraumbilical Hernia	Open Hernioplasty	NON-MALIGNANT	3.3	23.8	yes	wound infection
99	Vijayakumar	63/M	56288	Carcinoma Descending Colon	Left Hemicolectomy	MALIGNANT	2.3	17.7	yes	wound infection
100	Vimalabhai	55/F	1759	Carcinoma Sigmoid Colon	AR with end colostomy	MALIGNANT	3.6	22.1	no	

ABBREVIATIONS

BMI - Body Mass Index

SSI - Surgical Site Infection

MHMC - Mid Humeral Muscle Circumference

TSF - Triceps Skin Fold thickness.

DEXA - Dual Energy Xray Absorptiometry

TLC - Total Lymphocyte Count

PNI - Prognostic Nutritional Index

CI - Catabolic Index

DM - Diabetes Mellitus

BEE - Basal Energy Expenditure

BMR - Basal Metabolic Rate

RQ - Respiratory Quotient

ECM - Extracellular Matrix

CONSENT FORM

It has been explained to me in my mother tongue and I completely understand my condition and I have been explained in detail regarding this study - **"Pre operative serum albumin and body mass index as predictors of postoperative morbidity and mortality in elective major surgeries".** I hereby give my consent to participate in the above mentioned study.

Date:

Place:

Signature/ thumb print of the patient

with name:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது அறுவை சிகிச்சை துறையில் பட்ட பயிலும் மாணவன் அவர்கள் மேற்கொள்ளும் **"அறுவை சிகிச்சைக்கு முன் அளக்கப்படும் சீரம் அல்புமின் மற்றும் உடல் நிறை குறியீட்டெண் (BMI), அறுவை சிகிச்சைக்குப் பின்னர் ஏற்படும் நோயுறு மற்றும் இறப்பு விகிதத்தை அளவிட உதவுமா"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :